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# ROYAL COMMISSION ON MATTERS OF HEALTH AND SAFETY ARISING FROM THE USE OF ASBESTOS IN ONTARIO

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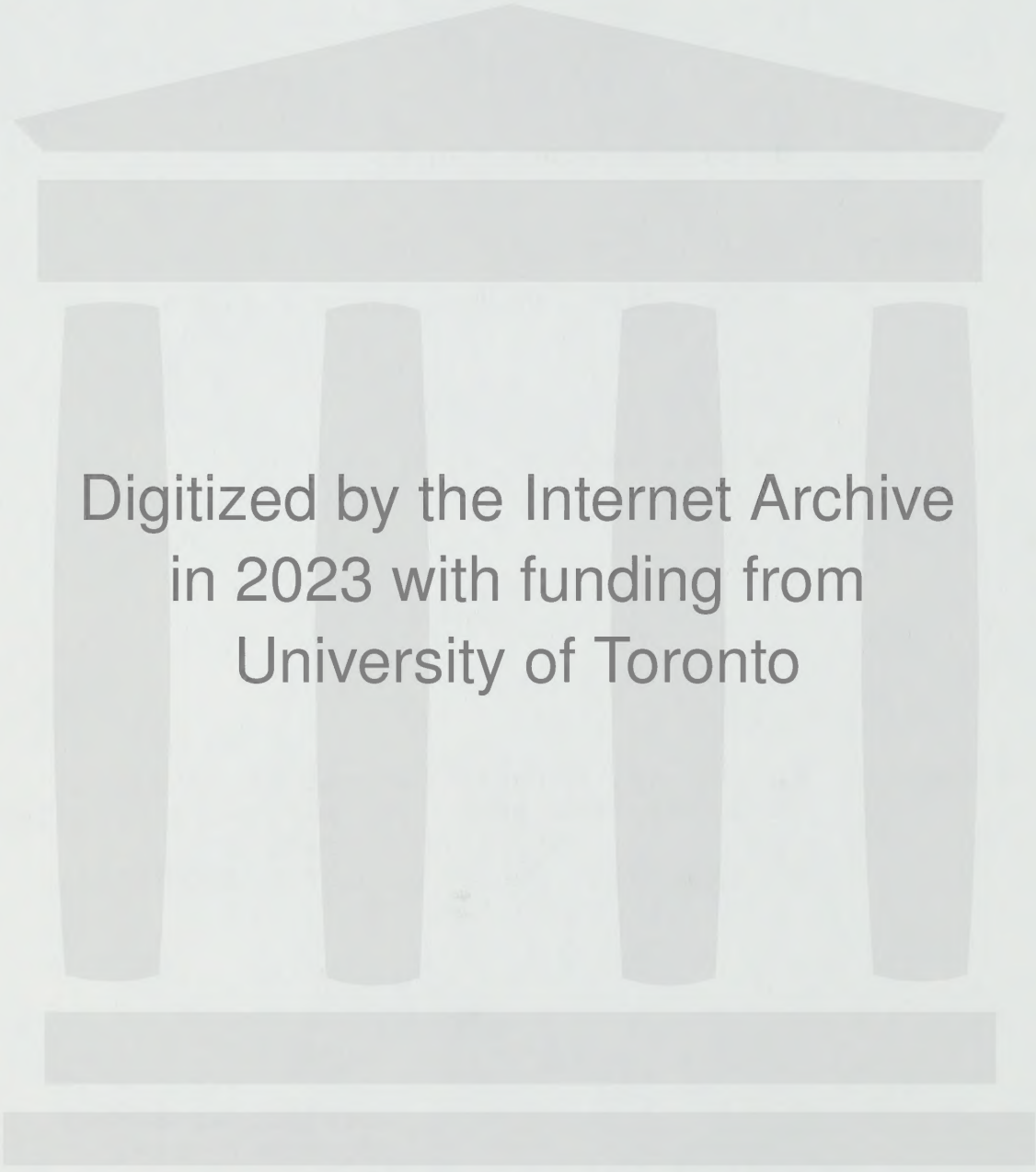
180 Dundas Street
Toronto, Ontario
Monday,
June 29, 1981

VOLUME XIV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ROYAL COMMISSION ON MATTERS OF HEALTH AND SAFETY

ARISING FROM THE USE OF ASBESTOS IN ONTARIO

VOLUME XIV

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THE FURTHER PROCEEDINGS IN THIS INQUIRY RESUMED PURSUANT TO ADJOURNMENT

APPEARANCES AS HERETOFORE NOTED

DR. DUPRE: Counsel, are we ready?

MR. LASKIN: I believe so, Mr. Chairman. I'm told that the people you see here are the people you have today.

DR. DUPRE: Thank you.

Well, may I first of all, on behalf of all of us, greet most warmly Dr. William Nicholson, who has kindly agreed to join us to give sworn testimony in what is really a course, Dr. Nicholson, what I am now referring to as epidemiology 101.

You are more than welcome in our midst, indeed, and we are very grateful to have you here.

Now, counsel, do you or your colleagues have anything to raise before I ask Miss Kahn to swear the witness?

MR. LASKIN: I don't, Mr. Chairman. I'll just canvass my colleagues.

I don't believe so.

DR. DUPRE: Very well.

Miss Kahn, would you swear in the witness, please?

DR. WILLIAM J. NICHOLSON, AFFIRMED

EXAMINATION-IN-CHIEF BY MR. LASKIN

DR. DUPRE: Please proceed, counsel.

MR. LASKIN: Thank you.

MR. LASKIN: Q. Dr. Nicholson, just briefly for the record, could you tell us what your present occupation is?

THE WITNESS: A. I am employed at the Environmental Sciences Laboratory of Mount Sinai School of Medicine, and have been so since 1969. There I am an Associate Professor of Community Medicine and Associate Director of the Environmental Sciences Laboratory.

Q. Could you just tell us briefly your educational training and background?

A. I have a Ph.D. in physics, with a minor in physiology and biophysics, from the University of Washington, which I received in 1960. My undergraduate degree is in physics, which I received in 1952 from MIT.

I was employed from 1960 through 1968 doing research in biophysics and solid state physics at Columbia University, and in 1969 went to Mount Sinai.

Q. I take it that one of your particular areas of interest has been the health effects of asbestos and asbestos exposure?

A. Yes. I have been doing laboratory and industrial hygiene work and epidemiology work on the exposures and the effects of asbestos in populations exposed to the fiber in different circumstances, as well as epidemiological studies of other groups exposed occupationally to different materials.

Q. Thank you very much.

We put before the Commission already exhibit nineteen, which is the red book that you see in front of you,

Q. (cont'd.) which contains some, but certainly not all, of your publications, and attached to it is tab eight of exhibit nineteen.

I understand, Dr. Nicholson, you have been kind enough to give us an opening address, an overview, as it were, of some of the issues, with the use of slide projector equipment?

A. Yes. What I'm going to do is just touch upon some of the issues that I would speak on perhaps in more detail as indicated by interest, that deal with the research that either I have been involved in or has been conducted at Mount Sinai School of Medicine.

I'm not sure...in addition to exhibit nineteen, which I know other people have had, I indicated there were two other publications, one of which is, the status as of yesterday, that I have supplied. I'm only asking if different people that are concerned have some copies of at least the one that is titled, Current Concepts in Occupational Carcinogenesis, because I would be referring to that in this brief introduction.

Q. It is being photocopied at the present time and will be down almost imminently.

A. All right. There is enough in the slides to deal with the situation until that time.

I guess if we could have the first slide...

Before going into some of the statistical data, which are table upon table, I first would like to just touch upon some historical items and then to also just bring the discussion to the reality of the disease experience, the real diseases that people are experiencing.

In terms of history, this slide has an historic significance in that it is a picture of a section of a lung, that was taken by Dr. Montague Murray in 1900 in Great Britain, and

THE WITNESS: (cont'd.) provided the information that he reported in 1907 at a compensation hearing, on the effects of asbestos on a group of textile workers that had been employed for approximately ten years in the late 1900's (sic) in which the individuals were virtually all deceased of what we now know as asbestosis, but at the time it was not properly recognized.

The dust conditions were egregious and at that time, though, some efforts to control them apparently had been made and Dr. Murray...the next slide...reported that, "One hears, generally speaking, that considerable trouble is now taken to prevent the inhalation of dust so that the disease is not so likely to occur as heretofore".

The hearing that he was addressing was one to determine whether asbestos disease should be compensated under the British compensation system, and with the wisdom supplied by Dr. Murray, it was decided that the asbestos diseases need not be so treated in Great Britain.

The previous slide showed the direct evidence of asbestos..if we could go back to it briefly for a moment...of asbestos exposure, in that the alveolar tissue was significantly fibrotic, instead of thin membranes between the air spaces there is thickened scar tissue. Additionally, there are numerous golden asbestos bodies, which are protein coated fibers. More careful microscopy, using electron microscopy, one would have seen numerous individual uncoated fibers as well, many more so than the coated ones.

Now, the subsequent slide...the other effects of asbestos exposure, beyond that of fibrosis of the lung, include plaques on either the parietal or visceral pleura, here shown on the parietal pleura at autopsy. These are not disabling, but are often diagnosed, largely diagnostic for asbestos

THE WITNESS: (cont'd.) exposures.

In addition...the next slide...one can have significant pleural thickening. In this section of a lung, instead of a thin membrane about the lung there is a one-inch thick pleural encasement, so with severe exposures not only do individuals have great difficulty in transporting oxygen from the air spaces into the blood stream, due to the scar tissue of the parenchyma, but they have great difficulty simply in expanding and contracting the lung, and death from suffocation is a very unpleasant way to go. These men often with severe disease have extreme difficulty. They walk upstairs backwards because they can sit down after every two steps in order to catch their breath.

But asbestosis is a disease that is of less importance compared to the malignancies. The next slide shows an x-ray of lung cancer which was actually first suggested to be associated with asbestosis exposure in 1935. It was not taken that seriously, no controls were considered or investigations undertaken to investigate whether this possibility was in fact a reality, and it was not until 1947 that it was determined to be so in a study in Great Britain of the British factory inspectorate, which found that thirteen percent of individuals deceased with asbestosis had lung cancer.

But even then when that was found in 1947, or in 1955 when a cohort study was published by Doll, regulatory action taking into account the carcinogenicity of asbestos was not undertaken. There is, I think, in the material that is being xeroxed, a paper that I published on the Regulatory Actions and Experiences in Controlling Exposure to Asbestos in the United States, which details some of the landmarks in disease identification from the turn of the century, and the accompanying regulatory action, and it's a dismal picture.

THE WITNESS: (cont'd.) Disease was found at many points in the past seven or eight decades, and little was done to effectively control it. And when control was taken, it was usually inadequate in that the thought was that by reducing exposures a certain amount, we will probably have eliminated disease.

But since the diseases that we are seeing often are the result of exposures decades previously, inadequate knowledge of exposures has limited our effective control because the exposures were ill-defined and the exact causal relationship not properly understood. The result of that is that the exposures have continued and workers are today seriously affected by our past lack of the full understanding of the problem.

The next slide, please. Oh, I'm sorry. Let's go to the next slide.

The other disease of most consequence for asbestos exposure is mesothelioma, which is the malignancy of the lining of the lung, quite different from the fibrotic pleura that was shown earlier. Here you see it encasing the lung of this individual at autopsy. It rapidly spreads throughout the chest cavity and in this case extending from apex to base. Because of its rapid spread the surgery is ineffective and to date chemotherapy is of little use other than prolonging a life a few months, and a very miserable life at that.

The next slide shows an individual with mesothelioma in the abdominal cavity. Peritoneal mesotheliomas, in fact, in some groups of asbestos workers account for more malignancy than does the disease in the pleura.

This, too, in the next slide, which shows a tumor in the abdominal cavity is widespread and rapidly-growing, and at time of diagnosis is invariably inoperable.

THE WITNESS: (cont'd.) Victims with both pleural and peritoneal mesothelioma are usually deceased within one year, or perhaps two, of diagnosis.

The next slide shows data that many of you have seen previously, and is the best data on the spectrum of disease that occurs from asbestos exposure. It is the analysis over a ten year period of time, 1967 through 1976, of the mortality experience of United States and Canadian asbestos insulation workers studied by Irving Selikoff of Mount Sinai.

The study is sufficiently large that not only are the common causes of death among insulators accurately determined, but so too are causes of less common...of cancer at less common sites. Cancer of the esophagus, larynx, pharynx, buccal cavity and kidney are all shown to be elevated compared to the rates that would be calculated...compared to the numbers that would be calculated using general population rates.

Overall, approximately sixteen hundred and sixty individuals were expected to have died. Twenty-two hundred and seventy-one did...about forty percent more than anticipated, and cancer was the dominant cause of the excess mortality, being elevated about threefold with lung cancer particularly a serious contributory cause of death. A hundred and five point six expected, whereas four hundred and eighty-six cases were found, based upon the review of all medical evidence, and I will come to that issue in a moment, the difference between death certificate diagnosis and that from a review of autopsy protocols, hospital records and surgical specimens.

In this slide particularly of interest is the fact that cancer of the kidney is elevated either on the basis of medical evidence or death certificate diagnosis. Eight cases found versus...I'm sorry...eight cases expected versus eighteen or nineteen found, depending on which data you utilize.

THE WITNESS: (cont'd.) The finding of tumors at other than the gastrointestinal tract or the lung where direct exposure to the fibers are clearly evident, comes about because the fibers can readily penetrate body membrane, can be carried throughout the body by either the lymph system or the blood stream, and be lodged in peripheral organs. In an analysis of various tissues by Arthur Langer at Mount Sinai, one can find asbestos fibers in virtually all body organs - the brain, spleen, kidney, liver, etc., and their presence there can be contributory to excess malignancy.

The next slide, please.

MR. LASKIN: Could I just identify that slide for a moment? I take it that's the same slide as table number one in tab nine, exhibit nineteen, your paper on Dose-Response Relationships?

THE WITNESS: Yes, that is correct. It is table number one of tab nine.

DR. DUPRE: It's table one, tab nine.

MR. LASKIN: Table one, tab nine.

THE WITNESS: Okay. Actually an extension of that same dataset, and I think it's contained in the monograph on Health Hazards of Asbestos Exposure, volume 330 of the Annals of the New York Academy Sciences, is shown here where cancer at other sites that are not common causes of death are shown... cancer of the pancreas, liver, bladder, testes, prostate, leukemia, etc. There, if one compares the data on the best evidence with that expected, there are not significant excesses of malignancy, although the entire group of cancers, other than the gastrointestinal tract and the lung, as a total classification is elevated at levels of significance, and this is probably the result of the dissemination of fibers to these peripheral organs, each of which has a risk...in which cancer occurrence is elevated but with the dataset that we have at this time, it is insufficient

THE WITNESS: (cont'd.) to say with ninety-five percent certainty that a given site is in excess. But I'm sure that in fact some of these cancers are indeed asbestos-related.

Now, on the issue...

DR. DUPRE: If the witness will just excuse me for a moment, as I look at table one in tab nine, that is not exactly the same thing.

THE WITNESS: Right. That table is not in tab nine.

DR. DUPRE: Okay.

THE WITNESS: That extends the data in table one by subdividing the category, All Other Cancers, according to the specific cancers themselves. In table one... let me just refer back to that...where is All Other Cancers... the All Other Cancer category was expected to contribute a hundred and thirty-one point eight deaths, whereas in fact it contributed a hundred and eighty-four. This is the excess of statistical significance that I mentioned before, with a forty percent increase based upon best evidence. Because the cancers are rare, each individual cancer as seen in this slide which now details the information according to individual site, does not achieve a level of significance, and the information is in this volume 330, which I think everyone has.

MR. LASKIN: Maybe I can...I think I can identify it more particularly, and everybody will know what it is. I believe it is from Dr. Selikoff's, et al, article, Mortality Experience of Insulation Workers in the United States and Canada, 1943 to 1976, and I believe it is table thirteen at page 105.

THE WITNESS: That's correct.

Now, let me speak about the difference between mortality experience compared using all information available on cause of death and that compared with death certificate alone.

MR. LASKIN: I take it BE is...

THE WITNESS: BE on this slide and the previous slide refers to diagnosis made using all available evidence, the best evidence available. DC is the information based upon certificates of death.

Normally in a study comparing the mortality experience of an occupational group with that of the general population, which here is that of the United States in that the insulation workers worked in all states, one would compare the death certificate causes of death with the rates in the general population, because the rates in the general population were determined by the cause of death as listed on the death certificate, and thus while there may be errors there, they would be equally likely in the general population as in the study group.

Unfortunately, with asbestos workers that is not the case, because two important causes of death among asbestos workers, asbestosis and mesothelioma, are virtually nonexistent in the general population, so the opportunity for misdiagnosis at those sites does not exist in the general population rates. Thus, to fully account properly for the asbestos-related mortality, one need look at all causes of death, particularly of those two sites. If you go to the previous slide that is table one of tab nineteen, one can see that one hundred and four deaths from mesothelioma were listed on the certificate of death, whereas one hundred and seventy-five in fact occurred.

Thus, seventy-one deaths from that cause would have been omitted had one simply utilized death certificate diagnosis. Similarly, asbestosis deaths on the death certificate accounted for only seventy-eight, but in fact a hundred and sixty-eight occurred. This is because those that were attributed under best evidence, not only were those listed

THE WITNESS: (cont'd.) as deceased from asbestosis or pneumoconiosis, but when corpulmonale, right-side heart failure, with asbestosis was the listed cause of death, it was attributed to asbestosis as it properly should be.

Now, if that was not done, then the alternative would be to have other causes of death be attributed to asbestos exposure when in fact they would not have the risk that would be apparent from death-certificate diagnosis.

The next slide, please, shows this. When one looks at...no, no....thank you.

When one looks at deaths from cancer of the pancreas, which happens to be a common diagnosis, a common misdiagnosis for mesothelioma of the peritoneum, on death certificates it was listed as a cause of death in forty-nine cases, so there would be nearly...had one used only death-certificate information, this would be unequivocal evidence that asbestos produces a threefold risk of cancer of the pancreas, and that would be a serious cause of malignancy among asbestos-exposed individuals, when in fact a review which removed twenty-six of those cases and attributed them to mesothelioma reduced the number to twenty-three, which is a slight excess, the proper perspective was seen.

When one finally looks at all this data, the one fact is that cancer was generally correctly diagnosed, there was maybe a five percent difference in the total number of cancer deaths attributed, and that's the important issue. The site characterization here is the proper one to use, but the argument that one should not count those based upon best evidence because you are not comparing like for like, is clearly improper.

I'm sorry. You were going to have a question.

DR. UFFEN: I just want to be clear about the

DR. UFFEN: (contd.) mesothelioma evidence, where the best medical evidence gave sixty-three....

THE WITNESS: No, the best medical evidence gave one hundred and seventy-five. It was a hundred and twelve of peritoneal mesothelioma, and sixty-three of pleural mesothelioma. The number one seventy-five I used referred to the combined cases.

DR. UFFEN: I think I want to be just a little bit meticulous about this as it had been stated previously to us that something like thirty percent of the death certificates had to be changed, they were wrong. I'm just trying to nail down whether that statement has any justification or not.

THE WITNESS: It's approximately correct. That is, one hundred and four death certificates listed mesothelioma as a cause of death. It was often just listed as mesothelioma with no site specified, but at least it was listed as a disease. In fact, a hundred and seventy-five were found, so that seventy-one additional cases of mesothelioma were attributed based upon all available evidence, which then allowed such causes of death as cancer of the pancreas to be put in proper perspective, rather than to be attributed as a very major cause of death amongst insulation workers.

Okay, the next set of slides...one more.

Among these, the study of Selikoff is also very useful in looking at the time courses of these diseases, because again of the large number of person years at risk.

Firstly, conditions have improved considerably from the turn of the century when Dr. Murray was reviewing the history of the last survivor of a group of individuals who had been employed, I think, a bare thirteen years previously, all of whom had died prior to his testimony.

Here we find that deaths from asbestosis among insulation workers generally do not occur until thirty years from

THE WITNESS: (cont'd.) onset of exposure, after which time there is a rapid rise in the risk of death so that observation of that disease is delayed long after initial employment.

The next slide shows the same data for mesothelioma. In terms of the death rate, it, too, rises significantly only after thirty years from first exposure. There are a few cases that occur in earlier years. This pertains both for peritoneal and for pleural disease.

The next slide shows the time course of lung cancer cases observed in this group of seventeen thousand, eight hundred men. The number of cases rises rapidly after about twenty-five years since onset of exposure, as seen in the upper curve. However, the relative risk between the number observed to that expected in the general population becomes significant sooner. I would like to spend some time now discussing the time course of the relative risk of lung cancer in different asbestos-exposed groups.

This is in the paper that I guess will be...is it now available or not?

MISS KAHN: It's supposed to be coming.

MR. LASKIN: Just before we get to that, are the last three slides reproduced in the publications, Dr. Nicholson?

THE WITNESS: Did I only use slides that are reproduced? It's similar to figure one of exhibit nineteen, tab nine, which lists the overall data on the number of cases versus time from onset of exposure, and figure two shows the relative risk of death from lung cancer according to time from onset of exposure.

The next slide...oh, sorry...shows material that is not in the tab, and that is what will be available. This slide is of lesser importance than the next one, which...but

THE WITNESS: (cont'd.) here I have plotted the data on the relative risk of death from lung cancer in insulation workers according to the time that the individual...in three separate groups according to the time that the individual was employed as an insulation worker. The black dots represent the data for those insulation workers that were employed first in the trade between ages fifteen and twenty-four years, the open circles for those individuals employed at ages twenty-five to thirty-four years, and the crosses for those individuals employed at age thirty-five or thereafter. The data in the lowest group are scant and are highly variable because of the small number, so I only want to focus on the data from the first two groups.

If you look at the curve, one sees that in each group it rises rapidly to a peak...in one case around age sixty, in another case around sixty-five...and then falls significantly at older ages.

But the two curves are separated by about a ten year period of time, suggesting that the relative risk of death from lung cancer is independent of the age at which the exposure began.

In other words, the slope, the increase in the relative risk, is the same for the exposure that began fifteen to twenty-five years as it is for the exposure that began ten years later at age twenty-five to thirty-five years.

Now, this is important. It's, the relative risk, in essence, does not depend upon the age. It has moved in age because there is a ten year period of time, and this comes about even though the absolute risk would increase two to four times for the group aged twenty-five to thirty-five compared to the group aged fifteen to twenty-four. Had the risk been independent, the relative risk been independent of the age, and in turn the...let me see, I'm sorry. Let me say it differently.

THE WITNESS: (cont'd.) Had the absolute risk, rather than the relative risk, been independent of age, one would then have seen a much steeper rise when plotted on these graphs. But because the two curves rise similarly, it is appropriate then to combine them and plot them according to time from onset of exposure, which is done in the next slide.

Can I have the next slide?

So that here are shown the same data that were on the other slide, but now plotted against years from onset of exposure, and what is being plotted is the relative risk - the ratio of the observed deaths to those expected in the absence of an asbestos exposure.

It does not matter, this same curve...the curve for, separately for the group that was employed fifteen to twenty-four years is identical to the curve on this graph for the group employed twenty-four to thirty-five years from exposure. They fall in the same line and thus the relative risk goes up identically for the young group as for the old group, even though the older group has a much greater risk that is being multiplied. So the absolute risk is much greater in the older group than it is in the younger group, but the relative risk...and its time course is the same.

MR. LASKIN: The absolute risk in the older group is higher than in the younger group?

THE WITNESS: Correct.

MR. LASKIN: Why? Because people are getting to a cancer age?

THE WITNESS: For the moment it is just sufficient to say that that is the observed facts, that is of the group that died who were employed...of the group that died zero to thirty years from first exposure and were employed from ages to twenty-five to thirty-five, more died of lung cancer.

THE WITNESS: (cont'd.) The extra asbestos-related risk was greater than that of the corresponding group who first were employed ages zero to thirty. But while that absolute risk was greater, the relative risk was the same and the time course of that relative risk was the same for the same exposure.

Now, I'll go a little bit more into the consequences of this.

DR. UFFEN: I just want to make sure that in the graph previous to this where you establish that the slopes were the same, was it zero to thirty years or was it...

THE WITNESS: No, that was...let's have the previous slide. The previous slide shows it versus age.

DR. UFFEN: And it goes from...

THE WITNESS: And they are shifted in...

DR. UFFEN: ...fifteen to twenty-five years?

THE WITNESS: Right. That was the age at which the person was first employed.

DR. UFFEN: Just a minute ago you used a sentence where you said zero to thirty?

THE WITNESS: No, no, I said...I was talking about the risk of death for the time zero to thirty years from onset of exposure.

DR. UFFEN: Okay. I was just confused for a minute that we had some fifteen year olds, from zero to fifteen year olds in that.

THE WITNESS: No, no, no. Mostly they began work at age eighteen.

But here you see the same time course of cancer as measured by relative risk for people first employed at different ages. So now, let's go back to the...yeah, thank you.

So what this slide in essence says, is that a given dose of asbestos will...whatever it might be...for this group that could be inhaled in five years, for example...will

THE WITNESS: (cont'd.) create a relative risk related to the dose of asbestos, and that as one adds additional doses an additional relative risk is added. Thus one sees an increase in time in this group of workers who are continuously employed. The asbestos effects that occurred from five years of employment, creating an increased relative risk, are added to by that of the next five years of employment. Thus as one continues employment one gets a continuously-increasing relative risk.

But it will continue to increase only if that which was created from the initial dose is then maintained at that level throughout a period of time at least equal to forty years, or to roughly age sixty. That is when the curve starts to go down. I want only now to focus on the rising portion of this curve. So that you have this...this curve is explainable with the concept that a dose of asbestos will multiply the risk of lung cancer in the absence of exposure, and will continue to multiply that risk in the absence of exposure for four decades thereafter, or until age, roughly sixty.

So it's a multiplicative effect with that which has taken place in the past and will take place in the future.

At age sixty or after roughly forty years from onset of exposure, when most of these insulators would terminate employment, one would have thought this concept would have had the curve remain flat thereafter. It falls significantly and this comes about because individuals that have been working and that are able to stay alive for whatever reason, smoking cigarettes, even, to age sixty-five in an asbestos environment have something that is different from the majority of them, and that is a survivor effect that is evident here. The exact details of it are not understood, but it's found in a wide variety of different occupational cohorts.

Those that manage to stay alive until age 65

THE WITNESS: (cont'd.) or thereafter have unique attributes that, if identified, might be of use in perhaps preventing some of this disease, but at this time are not fully known.

To some extent it is partly the result of people being removed from the group under study who are particularly at risk, as cigarette smokers. These would die both of lung cancer and of cardiovascular disease in the period up to forty years from onset of exposure, and thus the group would have a somewhat different composition after that period of time, and that can contribute partially to the fall. But that's not the full story. In fact, if you do it for cigarette smokers who were currently smoking in 1967, they also have a rise and a fall similar to that, indicating that it isn't simply the removal of cigarette smokers that is contributing to the fall.

Now, if you could pass that out I would like to focus on one curve in there to extend the same discussion.

MR. LASKIN: Would you just tell us exactly what this paper is?

THE WITNESS: Okay. What you are being...let me see what they are being given...what you are being given is a section of a paper that I have just completed as a contribution to a symposium held in Helsinki a couple of months ago, by the International Labour Organization, on occupational cancer. The full paper discusses some epidemiological data of a variety of carcinogens, including asbestos, and what you have here is the data and some of the discussion relating solely to asbestos.

MR. LASKIN: I'm just going to mark it into the record. Did you want to carry on describing it?

THE WITNESS: No.

MR. LASKIN: All right.

Perhaps in keeping with Mr. Warren's method of tabulating exhibits, let's call this tab eleven of exhibit nineteen.

EXHIBIT # 19, TAB 11: The abovementioned document was then produced and marked.

MR. HARDY: Can I, Mr. Laskin, ask a couple of questions about this exhibit? Namely, we don't have the full paper? We just have parts...

THE WITNESS: It's available here. I think it is only not being given for expediency at the moment.

MR. HARDY: We will receive the full paper later in the day?

MR. LASKIN: By all means, Mr. Hardy.

MR. HARDY: Particularly the references to even this section of the paper which we have, which is not included presently.

MR. LASKIN: As I understand it, what we've got is the portion pertaining to asbestos, and the corresponding graphs.

THE WITNESS: Right. It is the substantive data that I wish to discuss, relative to asbestos.

MR. LASKIN: Could I just make sure that I understand what you have just said about the slide which is before us, which I take it is...

THE WITNESS: Yes. Is there another copy of those data available so I can count the figures as well?

MR. LASKIN: It appears to be the seventh diagram which is before us, and...

THE WITNESS: Okay. Linda, that's okay.

MR. LASKIN: ...the slide before was the sixth diagram.

THE WITNESS: Yes. This slide is virtually the same data as the seventh diagram here, except on the diagram that you have in the text it does show the data for, the corresponding data for cigarette smokers in 1967, which indicates that the falloff is not simply due to the removal of smokers from the group...the statement that I just made previously.

MR. LASKIN: Dr. Nicholson, do I understand what you just said to be...and I'll put it in my own layman's terms and you can tell me if I'm right or wrong...but you are suggesting that every individual, apart from asbestos exposure, has a risk of lung cancer, be it smoking or the general environment or whatever, and when you add a dose of asbestos exposure that effectively multiplies that risk. The risk of lung cancer, independent of asbestos exposure, I take it, is going to be lower in people who are younger, rather than people who are older, so that when you add asbestos exposure to younger people, you multiply the risk but the absolute risk is going to be lower than if it were in older people?

THE WITNESS: Yes, that's correct, and I would just discuss that aspect now and it's easier to do with these data.

Firstly, just for reference, the first slide... no, no...no, the first, the first one.

Yes. In this paper the first graph, for reference purposes, shows you what the time course of lung cancer is in the general population in the absence of any exposure, as well as some other tumors. The point is that this is an extremely rapidly-rising function with time. In particular for lung cancer it's going up as the, approximately the sixth or seventh power of age. So what we are dealing with when we are talking about relative risks is a factor that will multiply a curve that is rising extremely rapidly with time, and that is why I made the statement that if one considers the absolute risk, that is the asbestos-attributable risk, of

THE WITNESS: (cont'd.) individuals exposed after age twenty-five compared to those exposed fifteen to twenty-five years, the number of cancers would be three or four times as many for the same period of exposure, because the risk that is being multiplied by the same factor would be three or four times greater...or the actual fact is, that observation of that much greater risk in the older people translates into the constant multiplicative factor that would operate independent of age.

Now, if one turns to the eighth slide or graph in this paper, the same information...and I'm sorry, I don't have a slide for this. That's why I'm glad we have this now.

The same information that I just referred to is seen in the study of asbestos manufacturing employees who manufactured amosite insulation during World War II and for a period thereafter in a plant in Patterson, New Jersey, and were studied by Seidman, et al, with the data being presented in volume 330 of the Annals of the New York Academy of Sciences. It's the article beginning on page sixty-one.

The data that are in this slide that you have here came from that article, although this slide is not in that article. But it shows conceptually the same feature.

MR. HARDY: Mr. Laskin, perhaps we should identify something about...

MR. LASKIN: Sorry. This is headed Lung Cancer, UNARCO, which I take it is United Asbestos and Rubber Company...

THE WITNESS: Right.

MR. LASKIN: The amosite plant.

Is everybody with us?

MR. CASGRAIN: I heard a sentence that the slide is in the book which doesn't contain the slide. Would you please say that again?

THE WITNESS: I said the data for this slide came from that article in the book, although the graph itself is not in the book. But the data, the exact data for this graph are available in that publication.

DR. UFFEN: Could you take a moment to explain... you've got relative risk, which we are accustomed to now, but you are also plotting relative rate. Rate of what?

THE WITNESS: Right..the rate of death from lung cancer. It's the number of cases per unit time...that is, lung cancers per hundred thousand person years at risk, so it's the absolute risk, it's the absolute risk of death, the absolute rate of death. It is not divided by the background risk.

Now, looking at the black curves, what one sees is that for a group of individuals who were largely exposed over a short period of time...ninety percent of them for less than five years, I think seventy-seven percent... ninety-some percent for less than five years. Seventy-seven percent, I remember, were exposed for less than two years.

So the exposure took place in one short period of time, and then the mortality experience was followed from five years through thirty-five years, by Seidman and Selikoff and others.

Now, looking at the relative risk of death at different years from onset of exposure, one finds that it is low for the first five year period of time, from five to ten years, and then immediately reaches the maximum that is achieved, a risk relative to that of the lung cancer rate in the absence of exposure, of five. Then it's maintained as a constant thereafter.

So that the exposure that took place for that group in that plant was sufficient to multiply by five times the risk of death from lung cancer in the absence of exposure,

THE WITNESS: (cont'd.) and to continue to do so for a thirty-five year period of time...even though the risk of death in the absence of exposure will rise nearly a hundredfold over that same period of time, which is manifest in the curve with the open circles.

Okay?

DR. UFFEN: Well, do you mind my saying, you treated the open circles data, you fit a curve to it?

THE WITNESS: I just drew a curve through that one.

DR. UFFEN: Yes, but on the closed-circle data, you eliminated the first point, although you drew attention to it, and then you drew a line through the rest of the circles, which is nearly a horizontal...

THE WITNESS: Right. Well...

DR. UFFEN: That seems a little curious to treat these two sets of data in a different way on the same graph.

THE WITNESS: Now, what we are saying is that it may take up to five years for the relative risk between...it may take from five to ten years for the relative risk that is attributable to a given asbestos exposure to be manifest.

DR. UFFEN: But why wouldn't you just draw... if you did that for the open circles, why wouldn't you draw one like that for the dark circles, and it would be...

THE WITNESS: Well, you would have left off all the points for the other ones.

DR UFFEN: Not if you drew it up through the center of them and weighted it.

THE WITNESS: Firstly, this one point consists of two or three deaths.

DR. UFFEN: All right. If it's not statistically significant, then you don't include it. But it's just difficult

DR. UFFEN: (cont'd.) for us to absorb the treatment of data at the same kind of the significance that you draw from it. But perhaps I can come back to it at a later stage.

THE WITNESS: Let's go back...

DR. UFFEN: You've left out the first point.

THE WITNESS: No, I really didn't. That first point does have significance.

Let's first...let me come back to it in a moment after going, again, back to this curve that you see on the...because the concept is an important one in dealing with the issue of latency, and I really don't want misunderstanding to occur.

Now, going back to this curve, which is explainable by the concept that each exposure creates an added relative risk for lung cancer, which continues to increase as exposure continues. If you draw a curve through the points as would best fit those points, a line labelled A is obtained. Then it goes off.

That line A, when extrapolated to a horizontal line equal to one, which is the risk in the absence of exposure, crosses that relative-risk-of-one line somewhere around five years, suggesting that the risk from the first exposure is manifest in about a period of five to ten years.

If you really stretch things to see how long it would take the relative risk to be manifest, the best you could do when you are really stretching it is to get a curve such as B, which I drew in such a way that you would still get most of the points, but the most that you could askew the curve to a crossing point at longer periods of time is one that would have it cross the axis of a relative risk equal to one at ten years.

So that this curve that you see in the graph,

THE WITNESS: (cont'd.) and this curve that you have in this figure here, each indicate that the relative risk from a given exposure is manifest within a period of time of about ten years or less. The point is low because, the first point on this graph is low because the time for the manifestation of that relative risk has not been fulfilled. After it has, which is up to ten years, then the curve rises to the full amount and stays constant thereafter. Here it continues to rise linearly thereafter.

So what we are seeing, actually, is...what I am illustrating is a concept different from the standard concept of latency where one would say that an asbestos exposure is not manifest for twenty or thirty or longer years.

In fact, the effects of that exposure can be manifest in a much shorter period of time, a period of time less than ten years. The manifestation is one in which the risk in the absence of exposure is multiplied by a factor relating to the dose of asbestos inhaled.

Now, if you multiply...if that exposure occurs in somebody aged twenty or twenty-five, the full manifestation of the risk would occur at age thirty, or thereabouts, but the risk in the absence of exposure is so low that you see no cancers resulting from it. But as the risk continues to multiply, the risk in the absence of exposure for decades thereafter, as that background rises according to the curve I showed you earlier, then the factor that you are multiplying becomes sufficiently great that cases then occur. Thus it is not until age forty or fifty that the lung cancers from an exposure at age twenty or thirty are likely to occur, and that's where we see them.

So in essence, the long lapsed period between an exposure at age twenty and a cancer at age thirty or forty really only comes about because the individual with the exposure is waiting for this background risk to catch up to a point where

THE WITNESS: (cont'd.) it becomes a sufficient amount that the multiplicative effect of asbestos then leads to a lung cancer, to a malignancy.

MR. LASKIN: Can I just try that in another way to see if I understand it?

I take it at, say age fifty or fifty-five, the background risk of lung cancer would be reasonably high absenting asbestos exposure?

THE WITNESS: That's correct.

MR. LASKIN: From smoking or whatever. So that if you have taken a person at that stage who has never been employed in an asbestos plant, never been exposed to asbestos, and then suddenly exposed them to a given dose of asbestos, whatever it may be, say at age fifty or fifty-five, under the theory that you are now advancing would you then expect to see a manifestation of disease within this five to ten year period?

THE WITNESS: Yes, and that's indeed what happens. Individuals exposed at older ages still have the relative risk manifest in this five to ten year period of time, but it's now multiplying something that is already very significant, and lung cancers appear in a very short period of time.

I will show you that, I think, in the next slide.

Yes.

MR. LASKIN: I've seen that table somewhere before.

THE WITNESS: Yes, that table is the table... it's from table nine of the article by Seidman, it's on page eighty-two of volume 330 of the Annals.

But first let me just focus a bit on some of the other information on that slide, which shows the relative

THE WITNESS: (cont'd.) risk of lung cancer during several ten year intervals, at different times from onset of exposure for different age groups.

If you look at the group aged forty to forty-nine, for example, there is the group that has higher exposure and lower exposure, and follow down that column in either category, you go for the five to ten year period of time with a relative risk of three point seven five to four point two seven to two point seven three. It's roughly constant, as is the risk in the higher-exposed group, which is now much higher because of more asbestos - it's eleven point nine four, eleven point four five, thirteen point thirteen.

That's the same constancy with time from onset of exposure that I showed you in the previous graph with the data combined.

Now, however, focus on the totality of data in each subgroup, the line labelled All, which is the analysis of the relative risks for all individuals in a category aged thirty to thirty-nine at onset of observation, forty to forty-nine and fifty to fifty-nine. So we are looking at the effects now for exposures in equal periods from onset of exposure. We are looking at effects at equal periods from onset of exposure in differently-aged groups, and that also is relatively constant. It's three point seven one to three point five two, and then it falls off - two point five eight, which is a reflection of this falloff at higher ages that I spoke of before. In the higher group it's eleven point one two, twelve point three two, and seven point four eight, and then falling off.

But you are seeing in the older people a manifestation very quickly of significant numbers of cancers, because the background rate is so high there. In the highest group there were eight lung cancers in the first ten year period of observation - nine times that which was expected.

THE WITNESS: (cont'd.) There was, for cancers in the forty to forty-nine year old group, eleven times that which was expected, and they were occurring in the period of time five to fifteen years from onset of exposure.

When you now look at the youngest group, age thirty to thirty-nine, because the background rate was so low there, there were no cancers seen in the period of time five to ten years from the onset of exposure. That's just a happenstance of that low background rate.

Had the risk of eleven point one two been manifest, one would have expected point six six cases to occur. So nothing occurred because the risk was so low in that decennial period of time, even though it was being multiplied by a factor of eleven.

MR. LASKIN: Can I just ask you while we are here, and it may be slightly away from it, does the intensity of the dose have anything to do with this analysis of latency?

THE WITNESS: No. I mean, what counts, the dose only is manifest in the amount of the relative risk. That is, a higher dose - a higher relative risk. I'll come later to dose-response relationships that would indicate that the relationship is a linear one. That is, you double the dose, you double the risk.

But the time course of the manifestation of that risk will be the same, independent of the magnitude of the risk.

MR. CASGRAIN: Say that again so that I can write it down?

MR. LASKIN: Do I take it the dose then affects the relative risk? The higher the dose, the higher the relative risk?

THE WITNESS: Right.

MR. LASKIN: But the dose itself does not affect the latency period independently of risk?

THE WITNESS: That's right. The curves that I would have described are the time course of the curves, are independent of the intensity of the dose.

But the curve where you had an increasing risk, if you had doubled the exposure the slope would have been twice as much, reflecting a doubled increased relative risk related to a doubled dose, so there is a dose-response effect that I will claim later will be linear - double the dose, double the effect.

But the time course of that increased risk will be the same.

What this...I guess I should mention this at this time...what this means is that an erroneous concept that I think got perpetuated by a guy named Hardin-Jones, who was studying radiation or something, that as you reduce an exposure you increase the latency time, and thus if you reduce it enough what you might do is increase the latency period to a time beyond the age of individuals, and thus you don't have to worry about low exposures and you have a virtual threshold due to a dose-dependent latency period.

That's wrong. There is not a dose-dependent latency period at all. What that is, is an apparent...what is happening is there is an apparent increased time to the appearance of cancer that is solely a dose effect.

If I can just use this...can everyone see this?

Consider the following situation: We are looking at lung cancer from exposure to, say asbestos. One would consider the lowest curve to be the lung cancer risk in the absence of exposure. Now, for argument's sake let's have a single massive dose applied at this period of time so we don't have to deal with a continuing exposure. It's like the

THE WITNESS: (cont'd.) UNARCO situation...the guy who gets a whopping amount in a year. Now, he gets a single unit of dose that will in future years produce a multiplicative effect on the lung cancer of one unit of relative exposure. I tried to have this be the same, but it's not quite. So that this in essence...this curve, in essence, doubles the background rate and proceeds with time like that. This is exactly in accord with the concept I have just been talking about.

A relative risk that is in this case equal to two, manifest throughout the lifetime of the individual...at least until age sixty.

Now, consider a second dose that is three times as great, it looks like, and thus this curve I am trying to draw is one that multiplies this relative risk by three times. It's just related to the arbitrariness of the situation.

But the point is, this is from a high dose, this is from a low dose, what I've drawn here is a constant relative risk for each of the two cases, one being three and one being two, which are multiplying this background risk. So this then would be the projected number of cancers seen at different periods of time, there can be a five-year lag period where nothing happens, as I've indicated previously. We are not looking at that period, we are looking now down the line.

Now, if you look at...

DR. DUPRE: Excuse me, Dr. Nicholson. What is the number of units of dose that you assume for your top line?

THE WITNESS: Multiplied by three...

DR. DUPRE: Your middle line you said was...

THE WITNESS: The amount of asbestos one inhaled in Patterson by working a year and a half.

I mean, it doesn't matter. I just...I want to talk about this business about a variable latency. It's just

THE WITNESS: (cont'd.) an arbitrary dose. This is more dose than that, but the multiplicative effect that I've drawn is one, that has the same constant muliplier throughout so that you get a widening separation here because you are multiplying this by three times compared to multiplying that by three times.

Now, if you consider a line, for example, where you look at the percentage of cancers that will develop in the population versus time, and say we chose five percent or ten percent where there is a real significant number...in fact until you get something of that order, you usually don't notice it because they are obscured in background..and you draw that line across here. One finds that it intersects the higher curve much sooner than it intersects the curve from the lower exposure.

So thus one would say that there is an increased latency in the lower group compared to that group, but that is not the case. All I'm saying is, the manifestation is sooner here because the risk is higher. You get the effect of the lower dose, but you don't get an increased latency, because I've not done anything about latency in drawing these curves. I have only dealt with dose.

In fact, the data that gave rise to this crazy thought was that of Drucke who did animal experiments with carcinogen..amino-something, I don't remember what it was... something that he added to their diets in different concentrations, and he added it in a variety of doses extending from a tenth of a part per million to a hundred parts per million...over a very wide range. He found that if you look at now the mortality according to time, that you get curves like that, with this being a high dose and this being a very low dose, and this is the percent cancer. In fact, the dose was so much that all the animals died of cancer very quickly. That is,

THE WITNESS: (cont'd.) these curves in fact went up to a hundred percent.

If you replot those curves according to the dose rather than time...well, let's leave that. Forget that last sentence.

What Druckery then did was look at the time that it takes to achieve a fifty percent...the time that it takes for fifty percent of the animals to get a tumor, and he got a curve like this. For the very low dose, it took a thousand days. For high dose, it took five hundred days, and so he got a curve that went like that.

So this is dose and this is time to fifty percent. The relationship was, the time was proportional to the one-third power of the dose.

That same relationship would be exactly the relationship one would expect if the time course of cancer were the third power of age, and so you get a curve going up for a certain dose that goes up as the third power. The higher dose, twice as high, would be increased.....just let me plot log of mortality in these creatures that we are talking about, versus age. For a very high exposure you would get a time...and that's what actually came from an analysis of data that he presented... that the time course of mortality increased about as the third power of age...I'm going to go the other way...a dose one half as much was a curve parallel down by just a factor proportional to the exposure, a lower one there, and this way the point at... any point along the curve was reduced by amount directly proportional to the reduced dose.

But then when you go across in this direction, the time to fifty percent tumors comes out to be exactly the one-third power of the dose.

So it's totally explained as a dose effect. There is no additional latency generated by the reduced exposure.

THE WITNESS: (cont'd.) You are seeing only the fact that the tumors are unlikely in earlier years, and they appear later.

Maybe there's questions at this point.

MR. LASKIN: I take it one of the theories which has been put to us, that time is inversely related to dose, is not a theory that... your analysis is different from that theory? In other words...

THE WITNESS: Yes. I would say it's absolutely wrong. There's places where people can disagree. I mean, this is higher, this is lower, but there really is...it's only due to the result of a wrong analysis that you would say that, from the data that have been even used to justify it.

Okay, I think that's...that which I have just talked about is the only thing that is significantly new from what either has been written here or is kind of standard knowledge in the field, so if we've gotten by this, the rest of it is...

MR. LASKIN: Relatively smooth sailing.

THE WITNESS: ...is relatively old hat.

DR. UFFEN: Can I ask a quick question? It is not about content, but since this is quite new and it's a bit new to us, too, has this yet...this hasn't been published? It's in the process?

THE WITNESS: It's in the process of being published. The manuscript was sent off this morning, and you have...

DR. UFFEN: It's still got to go through the peer-review procedure?

THE WITNESS: Well, no, it's being published directly in the proceedings of this conference. It is also being...will be submitted to...and that has yet to be reviewed... an article including this, as well as some dose-response data,

THE WITNESS: (cont'd.) we'll be sending it to the Journal of the NCR.

Okay, the next slide, please.

I just wanted to complete the picture of time course of cancer by looking at the corresponding data for mesothelioma. Here we can't look at relative risks because there is nothing in the general population to multiply the observed mortality rates for insulators. So the issue is that mesothelioma appears to be the...the absolute risk of mesothelioma appears to be related to onset of exposure, rather than the relative risk which is the case with lung cancer. This can be seen in the two sections on the right and the lefthand side of this graph, where I have plotted the data, again for those under age twenty-five compared to those over age twenty-five.

If you look at the age distribution where the data are kind of scattered, we are only dealing with a hundred and seventy-five cases, the absolute risk, the absolute death rate here is plotted rather than relative risk, and it is the absolute death rate now that is separated approximately ten years by age on the right side, implying that that is the quantity that one should then plot according to years from onset of exposure.

If you recall in the corresponding graph with lung cancer, it was the relative risk that showed this ten-year separation for individuals first exposed ten years differently.

When one does that, combining the two as in the middle slide, one finds that a very steep relationship, nearly T to the fifth power, obtains for nearly all cases of mesothelioma seen in insulation workers, and this now bears upon the consequences of exposure at very early ages. When you expose children, for example, in the circumstances in schools or other buildings, or in the home, because the death rate is going

THE WITNESS: (cont'd.) to rise so dramatically with time from onset of exposure and they have a long life expectancy, they can expect to have a very high risk for a given exposure, manifest relatively early in life and thus their overall lifetime risk will be considerably different from that of people exposed at older years.

This is different from lung cancer. There it doesn't matter too much what your age of exposure is. All the action is going to happen in later years when the relative risk from other factors rises to an area of consequence.

Here, with mesothelioma, it is rising independent of anything else that's happening to the individual, so the mesothelioma risks are much more important earlier on for earlier exposures.

DR. UFFEN: Can I just be sure of that? Now, this is a semi-logarithmic plot...

THE WITNESS: No, it's log log plot.

DR. UFFEN: Log log, all right. But that's an exponential growth...

THE WITNESS: No, it's not. It's a power log growth, because it is log log.

DR. UFFEN: In other words, you've got the times plotted as the logarithmic of the age along the bottom?

THE WITNESS: Yes, the bottom is a logarithmic plot. You can see it's like the difference between ten and twenty is nearly...

DR. UFFEN: Well, it's easy enough to see the left is logarithmic, but it's a little difficult to see that the bottom is logarithmic.

THE WITNESS: It is. The best you can see is the difference between ten and twenty is just about equal to the difference between twenty and fifty.

DR. UFFEN: In any case, it's not a linear

DR. UFFEN: (cont'd.) relationship at all.

THE WITNESS: No, and it's not even an exponential relationship. The data are unequivocal.

DR. UFFEN: It's a very high power, close to five.

THE WITNESS: Right.

DR. UFFEN: Which would mean that when you are older it's rising very much faster than when you are younger.

THE WITNESS: Yes, it becomes..it really is a serious thing at older ages.

Now, again what we don't have are data to indicate what happens after age forty-five. In insulation workers it tends to tail off. In fact I haven't plotted the last on this curve...I have it elsewhere...for the group aged fifty-five or over, which is somewhat lower than the highest point that you see there. This may be in part due to misdiagnosis of cancer in a population now aged seventy-five or eighty, because you have somebody exposed twenty to twenty-five, or there may be protective effects that are manifest in older people for mesothelioma as well. So I really don't know well what is happening after age, say seventy, here, or whether the curve even continues upwards after forty-five or fifty years from onset of exposure.

If it does, for somebody exposed at age five, it can be an absolute catastrophe, because continuing that curve upwards for...

DR. UFFEN: Five more years.

THE WITNESS: Five more years, is going to have very dramatic effects. We don't know what happens using data for insulation workers, after forty-five years from onset of exposures. The number of cases are few there, and the data are highly uncertain.

DR. DUPRE: Counsel, do we have that figure?

THE WITNESS: Even if you consider it flat, it's a catastrophe at that age.

DR. DUPRE: Do we have that figure in any of the current material?

MR. LASKIN: Perhaps with Dr. Nicholson's...

THE WITNESS: No, you don't.

MR. LASKIN: ...kindness, we might arrange to have a copy made of that slide so that we can distribute it.

THE WITNESS: Sure.

MR. LASKIN: Can I just make sure that again...

DR. MUSTARD: And the other table, counsel.

MR. LASKIN: I'm sorry?

DR. MUSTARD: The previous table I don't think is in anything either.

THE WITNESS: It's actually in this. That table is in this.

DR. MUSTARD: Do you want to give us a signal as to where it is?

MR. LASKIN: I think Dr. Nicholson referred to that previous table in one of the articles in the New York Annals publication.

THE WITNESS: The same data are in table nine of this, except I just reorganized them, and it is as seen there in the full copy of this, which you have a copy of. It's just that xerox we're viewing.

MR. CASGRAIN: Did you give the page earlier on? I didn't get the page number. You said it's page 310, is it?

THE WITNESS: No, no. Page 82 of the Annals.

MR. CASGRAIN: Page 82 of the Annals. But the difference below there, is that the Annals to which you are referring?

THE WITNESS: Pardon?

MR. CASGRAIN: Below that table, there is a reference...

THE WITNESS Yeah, that's the reference.

MR. CASGRAIN: Is that the actual reference?

THE WITNESS: That's the actual reference. The article begins on page 61.

MR. CASGRAIN: I see.

THE WITNESS: Now the table came from the data, which you have to do some calculations to get because you have to subtract stuff and..

MR. CASGRAIN: That leaves me out. I can't subtract.

THE WITNESS: I'm sorry. I think I misspoke. The table is table eleven. It's on page 84.

MR. CASGRAIN: Page 84.

THE WITNESS: If I said 82, that's the wrong table. That's referring to All Causes. But the table...

MR. CASGRAIN: It's eleven, not nine?

THE WITNESS: It's eleven.

MR. CASGRAIN: Page 84. The article starting at page 61.

THE WITNESS: Right. But you have to...one has to treat this cumulative probability that Seidman uses, which is... in doing this.

MR. CASGRAIN: Okay.

MR. LASKIN: Can I just make sure that, again, that I understand this? You are saying with respect to mesothelioma there is none of this background risk, non-asbestos exposed risk that we have, for example, with lung cancer, so that the critical matter is the time from first exposure and the risk of mesothelioma will certainly increase at least until you get up to thirty or forty or fifty years from time of first exposure, and therefore it matters whether your first exposure is at ten years of age as opposed to being first exposed when you are forty-five or fifty?

THE WITNESS: That's right. In other words, you

THE WITNESS: (cont'd.) shouldn't be exposed young and you shouldn't be exposed old.

DR. DUPRE: Let me see if I follow the second part of what you just said. I followed the first part in the dialogue between you and counsel, but did I understand you say that the second part of your conclusion is that you should not be exposed either at sixty or seventy?

THE WITNESS: Right. Well, no, let's make it forty or fifty. I don't know what happens to people, the risk factor at sixty or seventy. But when you are exposed and are a smoker, at age fifty, for example, you have such a high background risk for lung cancer that when you add the multiplicative effect of asbestos you will be getting cancer very quickly. So the proscription against employment at later ages is based upon the high risk of lung cancer that's very quickly manifest there. Proscription against young exposure is based upon mesothelioma.

DR. DUPRE: I just want to make sure I'm following you, because I ...

THE WITNESS: It doesn't matter for lung cancer...

DR. DUPRE: I'm just talking about mesothelioma.

THE WITNESS: Okay. The answer is no.

DR. DUPRE: This chart deals with mesothelioma?

THE WITNESS: That's right. And the age statement I made applies with the one that you shouldn't be exposed, particularly at young ages, because of concern for mesothelioma.

DR. DUPRE: Mesothelioma. Okay.

THE WITNESS: Okay, next slide.

Now, I would just like to go from this sort of discussion to a brief comment on some of the fiber concentrations that have led to the disease experience seen in insulation workers. These data are published in tab five. This slide, I think, is directly from tab five.

THE WITNESS: (cont'd.) It summarizes some of the concentrations measured largely by phase contrast microscopy, using the standard analysis method and personal sampling, by three groups in the United States.

MR. LASKIN: It's table seven, just so they can identify it, in tab five, page 164.

DR. DUPRE: Table seven in tab five?

THE WITNESS: In any case, at the University of California, at Harvard, and by us at Mount Sinai, would obtain very similar data for the average air concentration that asbestos workers were exposed to while in the course of using asbestos materials, and this took into account the different activities that they were engaged in, which might be mixing cement, applying the wet material, applying block around pipes or on turbines and boilers, or whatever.

The dust concentrations in individual activities were highly variable. If you measured for the two or three minutes that a person mixed asbestos cement, it could have a...one could measure a concentration of as much as a hundred fibers per milliliter, but then for the next hour when he was applying that wet cement there would be minimal dust in the air, and the average over that period of time was relatively low.

So that what you have is the major contribution coming from peaks of dusty activity, but the remarkable thing is that when concerned with the application of insulation, the air concentrations ranged from about three to nine, or somewhere with an average of about six. When the techniques that were characteristic of the trade were still being utilized, and before even the occupational safety and health administration had created a widespread awareness of the need for alternate methods. Based upon other data it would probably be the case that these exposures would be twice as great in earlier years because of a greater concentration of...I'm sorry, a greater percentage of

THE WITNESS: (cont'd.) asbestos in the product, so that we have estimated on the basis of the measurements made in the 1965/1969 period by different groups, that the insulation workers' exposure in land-based construction, which was largely the work they were engaged in, was perhaps between ten and fifteen fibers over the years dating back to World War II. In marine work this could be as great as twenty fibers because of the confined spaces and opportunity for greater exposure there.

Next slide.

Indicates one of the problems now with the measurement of asbestos dust concentrations, and that is that you are only counting those fibers longer than five microns, that are visible in a phase contrast microscope. If it's an extremely fine fiber, it's missed because it's not distinguishable, and if it's shorter it's not counted at all.

This...

DR. DUPRE: This slide, I gather, is the one that appears at page 157 in tab five, is that correct?

THE WITNESS: Yes. Oh, God, if I had known this was going to go on...

MR. LASKIN: I think that's right, yes.

THE WITNESS: Okay, 157 in tab five, yes.

I see what you are talking about. Everybody referring to...

MR. LASKIN: It's just helpful because when we go back to read the transcript of the proceedings, we will know when you are talking about something what we are referring to.

THE WITNESS: Right.

The fact that one enumerates only a very small portion of such fibers would be of no consequence, wherein the portion that's enumerated differs in different circumstances, so that we are not looking at a constant fraction of asbestos fibers in a variety of different work activities, we are looking at a

THE WITNESS: (cont'd.) highly variable one.

The next slide shows, for example, the distribution of fiber sizes during the fabrication of amosite blankets, and this is just a curve corresponding to that of tab... what is the tab?

MR. LASKIN: Five.

THE WITNESS: Tab five, but it does not appear in tab five. It illustrates that approximately thirty percent of the fibers are longer than five microns and thus counted.

I think, finally, going to tab five, table two, shows the percentage of fibers that we measured in a variety of such circumstances, and when the fibers are of chrysotile, the percentage varied from zero point four to five point nine. That is, there was a tenfold variability in the percentage of fibers that were counted by phase contrast microscopy. When one included the enumeration of amosite fibers, which are bulkier and larger than chrysotile, the variability was a hundredfold.

So what we have, in essence, is a rubber yardstick for the counting of fibers and much of the problems in trying to establish dose-response relationships and compare them between different work activities can come about from this variability. It can come about from other things as well, but there certainly are problems in actually quantifying work environment.

MR. LASKIN: Can I just make sure, and I think Dr. Uffen has a question, can I just make sure where we are? I take it we start from the proposition that counting fibers greater than five microns in length has always been considered just an index, because there are all kinds of smaller fibers, and you are suggesting that the proportions between the fibers smaller than five microns and the fibers greater than five microns may differ widely depending upon the product or the manufacturing operation, or whatever, that you are talking about, so that there isn't this constant proportion.

MR. LASKIN: (cont'd.) Do I also take it from the last slides that you are also suggesting that the variation may differ depending upon fiber type?

THE WITNESS: Yes, that's correct.

MR. LASKIN: All of that.

Is the proportion higher in one fiber type than another? I notice your amosite figures seem to be higher than your chrysotile figures.

THE WITNESS: Yes, the amosite is considerably higher than the chrysotile. We do not have corresponding data for crocidolite. From the data that does exist, it would appear to have a higher percentage of longer fibers than does chrysotile, as well.

MR. LASKIN: Is that something that you see relatively constantly, regardless of the particular industry or regardless of the particular phase of the asbestos industry? Do you generally see that the proportion of amosite or crocidolite fibers greater than five microns in length is greater than the corresponding proportion for chrysotile?

THE WITNESS: Yes, that's correct for amosite versus chrysotile, would have a generally...generally a correspondingly greater percentage count.

DR. UFFEN: Could I ask, could you give us some indication of the relative reliability of these data when you get down to the very small sizes, as compared with the ones greater than five microns?

THE WITNESS: Well, these data were obtained using electron microscopy, where all the fibers could be viewed. So the variability just depends more upon the number of fibers counted. I mean, this represented...they are clearly highly variable in one sense, but I don't know, maybe a thousand fibers were counted in each one of these studies.

DR. UFFEN: There would be no obvious trend or anything like that where it was easier to count the bigger ones? That's the point.

THE WITNESS: No, there might have...no, in general we are unaware of any systematic or significant systematic distortion there. This is simply an electron microscopic analysis of all fibers that were in view in a prepared specimen.

Next slide.

Oh, this should have been earlier.

But in any case, just to conclude on the issue of air sampling, there are different technical measures that are being proposed or suggested for the analysis of aerosols.

I'm not going to get into that because I haven't been that...haven't spent that much time looking or considering one method of counting versus another, and in one sense I don't think it's necessarily that important. It would have been important years ago had we been able to look at different situations where we now are seeing disease, to have that information, but unfortunately that time has passed and we no longer are capable of analysis of previous conditions.

One should be aware of the variability and to the best extent take it into account in analysis and in environmental monitoring that is done. But at this point one still needs a fairly simply method for evaluation of the work-place, and to go to very complicated...I'm not making the suggestion that one go to very complicated methods of analysis. I'm only making the point that one should be aware of the limitations of the simpler methods that we have at this time. And, if you can significantly reduce the concentrations compared to past exposures as measured by these simpler methods, you will correspondingly have benefited the worker who would have to work in such circumstances, so certainly one can effect controls without

THE WITNESS: (cont'd.) having the most sophisticated of measuring equipment available. And those techniques can also be used to monitor the continued efficacy of those controls.

The final point I would like to make with reference to this idea is the concept of the biological effects of fibers of different length. The five micron level has no significance other than it was an arbitrary size chosen...in fact it was chosen by the health physicist at the Turner Brothers factory in Rochdale because of the convenience and the ease of visibility of fibers longer than that length. The counters were asked, well, what should we count? He said, five microns and up.

It was a very good choice because most fibers five microns and longer would be at least a half a micron in diameter, and thus visible, and it was a convenient length for observation.

The data on the biological significance of fibers longer than five microns or shorter than five microns is very scanty. It's likely, and one could easily justify an argument that a longer and larger fiber has potentially greater health effects than a shorter and smaller fiber, if it reaches a site of importance. This certainly can even come about because larger fibers can break up into smaller fibers, so one big one can really equal two smaller when it gets to some point in your lung.

But because the smaller fibers are in such greater number, we cannot neglect their importance unless one has data that would indicate that they are at least one hundred times less important than the ones longer than five microns, because they are one hundred times more numerous.

And when you further consider that some of the biological effects that we see depend upon the penetration of fibers through body membranes, and their movement throughout the body, as well as their inspirability and deposition in the

THE WITNESS: (cont'd.) lungs, there the smaller fibers can proportionally have greater importance. They can be more easily...can more easily penetrate materials and be more easily carried about, so that while they may themselves, on a per-fiber basis, have less biological effect, their potential for getting to sites where their biological action can take place can be greater than the larger ones. So overall, between their number and their movement, they can in fact be more contributory to the disease that we see than are indeed those fibers longer that are counted.

But this is only a possibility. The data for this either way really don't exist.

Now, let's go...oh, this slide should have come just a little bit earlier, because I just wanted...oh, no, it's appropriate right now. I'm sorry. I know why I put it here.

Now I want to talk about standards because we are talking about the...I mean that's the subject of your deliberations, what are appropriate standards for workers.

Well, firstly, the standard that has existed in the United States and Canada, or various provinces of Canada, for a considerable period, for a decade at least, is a standard that is based solely upon asbestosis...the standard that was first proposed by the British Occupational Hygiene Society in 1968, which came from a review of x-rays of, I think, two hundred and ninety or two hundred and sixty individuals that were employed in a textile factory in the Midlands of Britain.

This is so, even though the disease of consequence is cancer and not asbestosis, as seen here when you look at the deaths of insulation workers. Of those occupationally-related deaths, there are six hundred and seventy-four excess deaths from cancer, compared to a hundred and sixty-two excess deaths from asbestosis. So what, four times as many deaths, occupational deaths,

THE WITNESS: (cont'd.) are from malignancy as are from asbestosis, and clearly that is the disease that one should focus upon in terms of standard setting.

But the initial considerations in 1966 and 1968 by the British, felt that their data for setting a standard on cancer were difficult and inadequate at that time, and so they didn't say that that was not an issue, but that they could not deal with that issue, in essence. Thus, they explicitly stated that their standard was for the prevention of asbestosis, and that standard was later adopted by the Occupational Safety and Health Administration, and in their deliberations in the hearings that led to the current standard in the United States, it was explicitly stated that great weight was given to the British standard that had been promulgated shortly before. Thus, the United States standard of two fibers per millilitre that we now have has a direct history going to the British deliberations in the late 1960's.

Unfortunately, not only was the disease of consequence not the proper one, but the measurement of the effects as seen in Great Britain was inaccurate.

The next slide shows information that was forthcoming in Great Britain in 1971, on the same population that was utilized for the observation of x-ray abnormalities in the consideration by the British of the status of workers employed in 1966. In an analysis of x-rays of those workers employed in the same factory in 1971, the then medical director, Hilton Lewinson, found at least five times as many individuals with abnormal x-rays as were considered to have such abnormalities in 1966. This is reflected in the data shown here, which is a slide I made many years ago to illustrate the inadequacy of our standard in the United States, even for the prevention of asbestosis according to the data that were then existing. The verification of the increased risk of development of x-ray

THE WITNESS: (cont'd.) abnormalities has subsequently come from data of Berry and others, that have been published in more recent years.

At the time that data were forthcoming, it was... other information came forth from Great Britain that would have suggested that the fiber concentrations were perhaps in error as well, and they suggested that the five to ten times greater prevalence of disease was matched by a corresponding misestimate of exposures in earlier years by five to ten times as much, so that the claim was that the overall disease experience for asbestosis might be somewhat the same.

I'm not sure about the accuracy of these latter day estimates of exposure, but the data that one sees in other circumstances on mortality for cancer would suggest that a standard of two fibers per millilitre, or a standard of one fiber per millilitre, is totally inadequate to protect workers, and I would like to focus on some data now on the relative risk of cancer in different occupational groups.

MR. LASKIN: Just before you leave that slide, do I take it from the heavy black line that what is being portrayed there is that at a hundred fiber years per cubic centimeter, which I take it over a working life of say fifty years equals a standard of two fibers, that somewhere between ten and twenty percent of workers will get some evidence of asbestosis?

THE WITNESS: Right, would have abnormal x-rays.

MR. LASKIN: That's morbidity and not mortality?

THE WITNESS: That's right. We are speaking of abnormalities characteristic of asbestos exposure. This slide was made before the suggested...before the British changed their fiber estimate that would reduce the concentration perhaps to two or three, according to their new estimates for which I have yet

THE WITNESS: (cont'd.) to see any data that would substantiate them. The only mention of them has been in the publication of the Asbestos Advisory Commission, or the British group that reviewed their standard and published the large volumes, which I'm sure you have here, and there they just give the overall data. You get factor of two because of the use of personal versus area monitoring, and another factor of two or three, vice versa, because of counting with a graticule rather than whole field counting. So that the two factors that they use to suggest a difference were mentioned, but there were no data whatsoever to indicate the accuracy of such an estimate or the basis for it.

The next slide.

DR. DUPRE: Just before you go to this next topic, Dr. Nicholson, I just wonder if I could put that slide and the table that preceeded it, plus your comment, in the context of what some of our earlier guest lecturers have shared with us.

Now, a point that has been advanced is this, never mind the standard...whether it's two, one, point one, point zero five, whatever...just thinking about standards in the abstract, a point that was made to us was that speaking in the abstract, a standard that was low enough to prevent excess mortality from asbestosis would automatically be low enough to prevent excess mortality from lung cancer. Now, is this something that you are bringing up? Counsel, am I...

MR. LASKIN: I think the proposition...I'm sorry to interrupt, Mr. Chairman...but I think the proposition was a standard low enough to prevent fibrogenisis, not mortality by way of asbestosis, but some fibrogenic dose, evidence of asbestosis, would also be low enough to prevent excess mortality from lung cancer.

DR. DUPRE: Right, not mortality from asbestosis. Right.

MR. LASKIN: In other words, a fibrogenic dose was roughly equivalent to a carcinogenic dose, and regardless of whether there was any medical evidence linking the two, if you prevented one you would prevent the other.

THE WITNESS: Well, I don't know what you are speaking of when you are speaking of a fibrogenic dose, whether you are talking about what would be microscopic evidence of fibrosis in an individual, whether you are talking about x-ray abnormalities of category one slash one or higher. You have a gradation there, and in fact with the evidence that is to date, it seems fairly clear that asbestos cancer is not...that asbestos cancer is present at doses that produce no visible fibrosis on x-ray. Where people would have normal x-rays, they have died of clearly asbestos-related causes, mesothelioma or lung cancer.

I mean, in such cases you might, you know, I don't know what the corresponding data is when one looks for microscopic fibrosis in all cases, for example, of mesothelioma.

MR. LASKIN: Would everyone like to take about five minutes before we go to the next topic? It might give you a break...

DR. DUPRE: Shall we reconvene at, say, twelve-ten by that clock?

THE INQUIRY RECESSED

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THE INQUIRY RESUMED

MR. LASKIN: I think we're ready to start again, Mr. Chairman.

DR. DUPRE: Please proceed, Dr. Nicholson.

THE WITNESS: Okay. I guess if we could have the slides again. Thanks.

MISS KAHN: This one?

THE WITNESS: No, no. The next one.

In the times that asbestos workers were exposed to the dust that contributed to their diseases, the respirators were generally unavailable to them on construction sites and so most individuals, many individuals, filtered the fibers through their cigarettes. The results of this have been especially serious.

The next slide shows...the next slide, one more... shows data that compares the experience of asbestos workers who did and who did not smoke cigarettes with corresponding individuals in the general population who worked in blue collar jobs and did or did not smoke cigarettes. For a detailed description of those general population workers, I guess go back to the slide that I skipped. The characterization of the controls are shown here, which were seventy-three thousand men in a prospective study of the American Cancer Society study, that had been enrolled since 1960. To correspond to the insulation workers, the ones selected were white, nonfarming, being exposed to dust, fumes and chemicals at work...that is, they were blue collar workers with a highschool education and they were alive on January 1, 1967, the date of the start of the observation of the insulation workers study that we have been discussing, of Selikoff's.

Prior to that study, he sent questionnaires to each of the seventy thousand, eight hundred men in the union, soliciting information on their current and past smoking habits, and from the questionnaires that were returned was able to separate, identify those individuals that had a history of cigarette smoking and those who had never smoked cigarettes.

The comparison now between the insulation workers and those men in the ACS study is shown in the next slide.

MR. LASKIN: Can I just identify that slide for the record, which I take it is taken from the article by Hammond, et al, Asbestos Exposure, Cigarette Smoking and Death

MR. LASKIN: (cont'd.) Rates in the 1979 Annals, and is table eight at page 487.

THE WITNESS: Right, table eight of page 487.

MR. LASKIN: Sorry, Dr. Nicholson.

THE WITNESS: For those individuals in the general population who were not exposed to asbestos and did not smoke cigarettes, the risk of death was eleven point three per hundred thousand person years. For those individuals in the general population who smoked cigarettes, the risk rose to about a hundred and twenty-two, or eleven times as great.

The corresponding data for asbestos workers were that nonsmoking asbestos workers had a fifty-eight...had a risk of fifty-eight deaths per hundred thousand, or five times that of the nonsmoking controls. And insulators who did smoke cigarettes had a risk of death six hundred per hundred thousand, or fifty times the individuals who did not smoke nor work with asbestos.

What is happening is that the two risks, the fivefold risk of exposure to asbestos in nonsmokers, and the ten or elevenfold risk of cigarette smokers in the absence of asbestos exposure, multiply together to produce this enormously increased risk. It in fact is a specific reflection of the data that I was presenting previously, that being that the effect of an asbestos exposure of a given magnitude is to multiply whatever the risk is in the absence of exposure.

For insulation workers the effect is such as to create on average a fivefold multiplicative risk whether for nonsmokers or for cigarette smokers, but the consequences for cigarette smokers is far more dramatic because you are there multiplying a considerably increased risk.

The other factor is, and here I will refer to the article by Hammond, in the Annals...the same article you mentioned...if I can find it.

THE WITNESS: (con't.d) Where if you look at individuals who have ceased smoking cigarettes, the effects are correspondingly reduced. That is, if you alter what would be the background risk, you get a correspondingly altered benefit. This comes about because if you look at the mortality rate for lung cancer in individuals who stop smoking cigarettes, one finds that it can fall dramatically over ten years to perhaps a quarter or even less than that which it would have been had one continued whatever the cigarette habit would have been previously.

For asbestos workers, referring to page 483 of Hammond in the Annals, where table 6 B shows the mortality ratios for individuals who ceased smoking in different periods of time...and I will just read the data for those individuals who were smoking, at one time, twenty or more cigarettes per day.

For individuals that continued to smoke, and this is for comparison purposes, the ratio of lung cancer to that in the absence of cigarette smoking was ten point four. That's roughly equivalent to the data here, but for the specific subset of twenty-plus cigarette smokers, the ratio was ten point four. It was slightly less for those otherwise.

Wait a minute...just a minute. Oh, these ratios that I'm giving are ratios of the risk of death in insulation workers to insulation workers who never smoked regularly, so we are comparing now insulation workers with insulation workers, not with the general population. But the same tenfold risk applies insulation workers to insulation workers, as does controls to controls as you see there.

Now, for individuals that ceased smoking less than five years ago, the risk of death from lung cancer is eleven point five. That is, it's higher than those that continued to smoke. This comes about because many people in that category who had ceased smoking within the last five years did so because they started coughing up blood and had a diagnosed

THE WITNESS: (cont'd.) lung cancer, and thus mortality...their reason for stopping was the finding of disease and that led to a distortion of the data. But thereafter, for those that ceased five to nine years and ten or more years ago, the ratio dropped to four point one five and three point three nine.

In other words, after five years of cessation of cigarette smoking, the risk of death from lung cancer decreased to perhaps one-half or one-third what it would have been had one continued to smoke cigarettes. So there is considerable benefit that can be occasioned by cessation of that habit, and certainly even more benefit by never beginning it.

Okay, that's cigarette smoking. Let's go on to the next slide.

Now, I would like to turn...in fact, leave the slides off.

I would like to now spend a little bit of time in discussing dose-response relationships in a variety of studies, and here to speak directly to some of the, using some of the data that have been presented in tab nine, exhibit nineteen, and in particular focussing on tables...or figures four, five...figures four through ten, and most importantly, on table...

MR. LASKIN: Fourteen, I think.

THE WITNESS: Fourteen, I believe. Table fourteen.

As I mentioned earlier in the discussion of the time course of cancer, I stated that there was a dose effect and you would have that reflected in the different data that I showed by either increasing or decreasing the effect as manifest on the slides, shown by the increase or decrease in the total amount of asbestos inhaled.

The justification for such a statement that

THE WITNESS: (cont'd.) the risk of asbestos is proportional to the dose is seen at in at least four studies that provide data on the relative risk of death from lung cancer, and for all asbestos-related diseases, according to the exposure, the cumulative exposure of the individuals under observation.

These four studies are the study of Asbestos Factory Employees, by Seidman and others, and published in the Annals of the Academy, New York Academy of Sciences, beginning on page 61; the study of Retirees of an Integrated Asbestos Products Manufacturer in the United States, by Henderson and Enterline and published in the same Annals beginning on page 117; the Analysis of Data on Canadian Asbestos Mine and Mill Employees largely described in the paper of Liddell and others, published in the proceedings of the Royal Academy, or Journal of the Royal Society, volume 140, page 469, 1977; and in the data presented by John Dement at the Fifth International Conference on Inhaled Particles, last September in Cardiff, Wales, and to be published in the proceedings of that conference and available as a preprint from Dement as the paper and as well in his thesis.

The data from these studies are shown in the slides that I gave, and over the doses observed one finds a linear dose-response relationship to obtain in all cases.

If you let me leave the data for Seidman, figures four and five to the last...looking at figure six, the risk of lung cancer and of all asbestos disease, which would include asbestosis, lung cancer and gastrointestinal cancer, and kidney cancer in the case of Seidman, et al, but I don't think it contributed much at all...but largely gastrointestinal and lung cancer, one finds a linear relationship with dose.

So, too, in the study of Enterline and Henderson in figure seven, and in the data of McDonald and Liddell and others in figure eight.

MR. LASKIN: Could I just ask you two questions at this point, just so that I'm clear?

THE WITNESS: Mmm-hmm.

MR. LASKIN: Are you saying that the data which you just talked about in the studies you have just talked about, demonstrate a linear dose-response relationship or are compatible with a linear dose-response relationship? Because we've had, for example, some evidence from, for example Mr. Berry, who put forward the proposition that his data would be compatible with any number of...

THE WITNESS: He was looking at x-ray data, was he not?

MR. LASKIN: Leaving him aside, do you say that they are simply compatible with the linear dose-response?

THE WITNESS: Yes, I guess you really have to say they are compatible with, because the problems that...there are several problems that exist in these studies. Firstly, some of them are...there is the issue of dealing with average exposures for a group and categorizing people according to that average exposure. Firstly, sometimes the average exposure is low because of short-work employment, rather than due to exposure to lower concentrations, and the measure of exposure being years of employment. Often people with short-term employment get lousier jobs and that might not be properly reflected in their average exposure.

The time course of the exposure over many years may not be accurately measured and estimates made of exposures in later years may not correspond to those for the same job in earlier years. So there's a lot of problems with that.

Another problem that comes about is that some of these studies are, that of Enterline, for example, and McDonald I know...I'm not sure, it doesn't have that much effect for Seidman because all the exposures were short in duration.

THE WITNESS: (cont'd.) But those exposures categorized an individual according to the cumulative dose that he received, which means that the person years that he would have accumulated in that study, and thus the determination of the denominator, was attributed to the highest exposure category that he had, and not to each exposure category that he passed through on his way to achieving his ultimate exposure. So that you then have a very high denominator in a high category, and a lower denominator in a lower category, which tends to depress the observed dose-response relationship, and to some extent perhaps also distorted in terms of linearity in that.

So there's a whole bunch of complicated things that go into these things that could preclude one making an absolutely firm statement that there is no question of a linear situation, and I don't want to do that.

MR. LASKIN: Can I put three statements to you and you tell me whether one, two or all three are fair or what is the matter with them?

The first statement is that a linear relationship fits the epidemiological data as well as anything else.

THE WITNESS: That's absolutely true. The linear relationship is the best relationship that we have at this time, although the data that exist would indicate that it may be too conservative. That is, the data, if anything, on an epidemiological basis, is just...what we see there appears to indicate that lower exposures and shorter exposures may have greater effect than you would predict on the basis of a linear relationship.

MR. LASKIN: More than proportional?

THE WITNESS: Right.

MR. LASKIN: All right. Well, leading up to my second and third propositions, which are: Is it also fair that this linear relationship, at least on the basis of the

MR. LASKIN: (cont'd.) epidemiological studies which you have looked at, the line will go through zero?

THE WITNESS: If it is allowed...if one fits a curve with no requirement that it go through zero, some of the studies would have the line go through the origin at a finite risk. What does not happen in any study is that the line goes through the risk of unity, that is the risk in the absence of exposure, at a finite exposure. So that the deviation from a line allowed to, fitted with no boundary conditions would pass through the axis at higher relative risk, rather than the axis of higher exposure.

MR. LASKIN: I'm not entirely sure I understand that.

THE WITNESS: I guess it's best seen in the data of...what I'm saying is best seen in the data of Seidman and others, where you see from the curve that I drew which I forced to go through the origin...that is the origin in dose, at a relative exposure of one, that is the point expected in the absence of exposure. There are data points above that line.

If the requirement that it pass through one were not made, and in fact when I did a regression analysis with no boundary conditions, a curve approximately like that would be drawn, which would have it pass through the origin at a...pass through the origin in dose at a relative risk greater than one.

If you look at each of the curves I am describing, in none of them would it, for example, pass through the origin at a relative risk less than one. That is, you never find a curve allowed to be fitted with no proscription pass through the origin of dose down at a relative risk less than one, which would be suggestive of a threshold.

MR. LASKIN: All right. That's really what I was coming to. Are you saying that from your analysis of the

MR. LASKIN: (cont'd.) data you are not forcing any of those lines through the Y intercept at a relative risk of less than one, which would imply a threshold. In other words, you are saying that the studies support the proposition that there is no threshold effect, as it were?

THE WITNESS: Yes. None of the studies give any evidence for a threshold effect of asbestos exposure, and all of the studies are best described by the use of a linear dose-response relationship, although in some cases an even more severe relationship...that is, one that would have increased effect at lower exposures...may obtain.

MR. LASKIN: The third statement, and you may be coming to it later, is simply this: I take it that many of your observations are at higher exposure levels and to some extent there is an extrapolation along this line? I'm assuming that's fair. I am just wondering whether in your professional opinion it's fair, at least in terms of an approach to standard setting, to extrapolate along the linear dose-response line which goes through the Y intercept at one?

THE WITNESS: Well, for those studies that we don't have a succession of points at different doses, that's pretty much what we have to do. There is no...the data of the four studies would suggest that that's appropriate and in particular that's what I have in fact done in those studies for which the data have been inadequate to really do an analysis over a full range of doses.

MR. LASKIN: Thanks, Dr. Nicholson.

THE WITNESS: In any case, firstly with curves such as are in figures four through ten, one has a measure of the...one has several measures of the effect of various doses of asbestos in different circumstances, and these measures differ considerably. They have been, as best as possible, translated to

THE WITNESS: (cont'd.) a common measure by estimating the dose of asbestos in these circumstances in terms of fiber years per millilitre, even though the measures that we have may have been in terms of other exposure indices. In particular for mining and the asbestos products manufacturing, and the exposures of Dement, the initial measurements in the years distant, in the distant past, were in terms of the total number of particles per cubic foot where all objects visible in a light microscope were counted.

As variable as the fiber counting is, when you now add counting of all dust particles in the workplace, you've got much more variability. And when you consider in the manufacturing operations that many products were, consisted largely of dusts other than asbestos...calcium silicate in terms of insulation, talc with other products, silica dust and whatnot... and these dusts were also being counted, you can have fairly severe distortions of the air concentration.

Some measurements have existed where...have been made side by side of dust concentrations using fiber counting and particle counting. These have been mentioned in the asbestos mining and milling, and McDonald has stated that he feels that an average of...let me just make sure, two or three...

MR. LASKIN: I think three.

THE WITNESS: Three? All right. Whatever he said, I used.

MR. LASKIN: In fairness to him, last week he indicated some doubt as to the reliability of his conversion factor.

I believe you used three.

THE WITNESS: I used whatever he said. I just had forgotten that number.

Dement provided the most data on the

THE WITNESS: (cont'd.) convertibility between fiber counts and particle counts, in that he had something like eleven hundred side-by-side or concurrent measurements, and these yielded a factor of ...

MR. LASKIN: Three as well, I think.

THE WITNESS: Yes. Showed that three fibers per millilitre were equivalent to one million particles per cubic foot, with a fairly narrow confidence interval of two to three point five, and a value of eight characterized fiber preparation work which only a few people were employed in, and he, Dement used these conversions to translate his data into estimated cumulative exposures of fiber years per millilitre, and in fact that is what is displayed in the graph on figure six, but unfortunately the graphics person left off the bottom axis which is, as all the others, in fiber years per millilitre.

The data for Seidman, the estimates for Seidman came from membrane filter measurements made in plants manufacturing the same materials to the same specifications with the same equipment that were operating in the early 1970's...well, from 1968 through 1972. The plants involved were operating with very limited controls, one with hardly any at all, and was in a low, double-quonset building with conditions as dusty as any I've ever seen, and were unlikely to be worse than those of the Patterson plant, which was in a rather large building, and the comparability between these two other plants and the fact that it's kind of difficult to keep a dust concentration of much more than forty or fifty fibers in the air, would suggest that that's an appropriate measure for the average exposure of individuals employed in Patterson, and thus I used average exposure of all samples in those two plants as measured by NIOSH to convert period of employment in the Patterson plant to an estimated cumulative dose of fiber years per millilitre, the value

THE WITNESS: (cont'd.) being thirty-five, I believe, fibers per millilitre as the average exposure.

Some people would have higher, some people would have lower, but that was the average exposure, the average concentration of all samples made in Tyler, Texas, and Port Alleganie, Pennsylvania.

Thus, with such estimates of exposure one can calculate relative risks per unit dose from the slopes of the lines in the different graphs. These data are plotted...these data are listed in table fourteen for the four studies that I just mentioned and for other studies for which there are average estimates of exposure for groups of individuals such as insulation workers, where I indicated an average exposure of about ten to fifteen fibers was felt to characterize their exposure in earlier years, and with a twenty to twenty-five year exposure time obtained a cumulative dose that would be attributable to the disease experience we now see.

MR. LASKIN: Can I just make sure I understand this by going through one of these calculations with you, to make sure I understand it?

THE WITNESS: Sure.

MR. LASKIN: If we take the amosite insulation manufacturing establishment on table fourteen...

THE WITNESS: Okay.

MR. LASKIN: ...and go back to figure four.

THE WITNESS: Right.

MR. LASKIN: Do I take it if you draw a line out from the relative risk of two on figure four...

THE WITNESS: Right.

MR. LASKIN: ...along the slope of the line, and we draw then parallel to the Y axis, you will get a relative risk of two at about eleven fiber years per millilitre? Am I accurate so far?

THE WITNESS: Right.

MR. LASKIN: If you then, to find out what the percentage risk is at one fiber year, if you divide the eleven into a hundred percent, which I take it is...

THE WITNESS: A hundred percent, which would occur for eleven.

MR. LASKIN: Right. A hundred percent is equal to a relative risk of two, it's a doubling?

THE WITNESS: Right.

MR. LASKIN: So you divide eleven into a hundred and you get a risk of...you get a percentage risk of nine point one for every fiber year per millilitre or per cubic centimeter?

THE WITNESS: That's correct.

MR. LASKIN: Leaving aside whatever inaccuracies or accuracies, or whatever methodological limitations there may be, if the thrust of that figure means that at a...does that mean that at a one fiber standard over some average working life you've got an excess risk of lung cancer in amosite insulation workers of nine point one percent, or not?

THE WITNESS: If you work for one year at a fiber per millilitre, there would be an excess risk of nine point one percent in these circumstances. Or if you work for ten years, you would roughly have..you would nearly have a doubling.

MR. LASKIN: You have a doubling of the risk at one...

THE WITNESS: Fiber per millilitre.

MR. LASKIN: I see.

THE WITNESS: According to these data.

In terms of...I should mention...in terms of the issue of...and in Seidman's data, this is the only one that has a large number of points significantly above the line...if

THE WITNESS: (cont'd.) one had used simply the slope of the line without the constraint that it pass through one, you would have a slightly reduced slope. That slope would be roughly three percent per fiber year per millilitre, so then employment for thirty years at one fiber per millilitre would lead to a doubling of the lung cancer risk...and with the same analysis, an increase in all mortality by ten percent.

MR. LASKIN: Just a point of clarification on all mortality, when you use the phrase All Asbestos Disease, does that include mesothelioma?

THE WITNESS: Yes. Mesothelioma, lung cancer, gastrointestinal cancer and asbestosis.

MR. LASKIN: Are you suggesting that all asbestos disease also has a linear dose-response relationship with asbestos exposure?

THE WITNESS: It would appear to be roughly so. The dose-response relationship for asbestosis is somewhat different from that of lung cancer and mesothelioma in that deaths from asbestosis do not occur at low exposures. There really is like a threshold for mortality from asbestosis.

The data on the dose-response relationship for mesothelioma are very scanty. We have very little information there because the only study that has...the studies in which one looks at relative exposures, such as in Seidman, have very few deaths in the different subcells. Those data that do exist are completely consistent with a linear dose-response relationship for mesothelioma as well, and logic would have such a relationship...would have one use such a relationship analogous to what we find for lung cancer. But it is not...the substantive data are not there as they are for lung cancer.

MR. LASKIN: Looking at...can I just ask you this, and you may be coming to it in your talk, but looking at table fourteen, of course, one of the things that interests

MR. LASKIN: (cont'd.) this commission is the differences that we see. Throughout the course of our testimony so far we've had various hypotheses and explanations put forward as to what might account for these differences. For my own purposes, I have identified six different factors and perhaps I can just put them to you at the outset and you can...I would appreciate your comments on them, and if there are some additional factors or explanations, I would appreciate your comments on those.

But let me list the various factors for you:

Number one, a difference in fiber type as between amphiboles on the one hand and chrysotile on the other.

Second...

THE WITNESS: Do you want me to comment as you go along?

MR. LASKIN: Why don't I list them all for you, and we'll come back to them.

Second, a difference in fiber size distributions as asbestos goes through various processing operations and so on, from mining through to manufacturing, and so on.

Thirdly, the hypothesis that a cocarcinogen may be involved in the later stages of the asbestos process that may be interacting with asbestos.

Fourth, that the intensity of the dose may be an important factor in the sense that you may get different health risks if you get the same amount of dose in less period of time. For example, Enterline hypothesized that may account for the high hazard amongst his maintenance workers.

Fifth, that there is somehow a change in the physical state of the fiber itself as it leaves the mining operation, for whatever reason.

Finally, that this is kind of a catch-all category in which I include a number of things, but the methodological differences and limitations and so on that may

MR. LASKIN: (cont'd.) be evident in a number of these studies...and by that I mean everything from exposure estimates, fiber conversions, time of observation periods, the accuracy of ascertaining the cause of death, the proper measurement of dose.

Those are six things that I've tried to identify and I certainly would appreciate any help you can give us on those, so can we go back to the beginning and start with the whole question of fiber type?

THE WITNESS: Yes. I think when you are making a comparison between fiber counts and disease, fiber type is important because as I showed earlier, the counting of asbestos fibers enumerates quite a different quantity than does counting of chrysotile fibers, and they are different in shape. On a per-fiber basis, that different size distribution in the different percentage of it counted would seem to suggest that differences in relative risk per fiber per dose, in terms of fiber years per ML, could exist. It would appear that in fact it does. For example, the highest here occurred in circumstances where some amosite exposure was present, in the data of Seidman and to some extent in the data of Newhouse and perhaps, to some extent, with insulation workers. Although at the time that we were making these measurements, the measurements were largely of chrysotile because the use of amosite in insulation had begun in the 1940's, and was significant in the 1950's, but in the 1960's was pretty well phased out.

The exposure to amosite is a relatively unknown factor there.

The remarkable thing, in fact, is that the estimates of the two fiber types are even reasonably close in terms of...we see, for example, high levels with amosite, or we see virtually the same high level in the textile production study of Dement et al, where a risk of...we just call it risk

MR. LASKIN: The females in Newhouse and Berry.

Just so that I understand it, if we use Dement by way of example, a half a fiber over forty years equals, I guess, twenty fibers, and if you multiply the twenty by five point three percent you get a hundred percent, doubling of the risk?

THE WITNESS: That's right. Yes, that's right.

MR. LASKIN: It might be a convenient time, Mr. Chairman, if we broke for lunch and gave the witness a little rest.

DR. DUPRE: Our customary hour and fifteen minutes, and we'll reconvene at two-twenty.

MR. LASKIN: I think that's appropriate.

THE INQUIRY RECESSED

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THE INQUIRY RESUMED

DR. DUPRE: Counsel, are you ready to proceed?

MR. LASKIN: Thank you, Mr. Chairman.

Dr. Nicholson, we have been discussing tab nine of exhibit nineteen, and just to make it clear, because the tab nine that we've got is a, appears to be a draft document, but as I understand it, this document in its final form formed a criteria document for the government of Sweden in respect of its discussion of occupational standards for asbestos exposure?

THE WITNESS: Yes, that's correct.

It's published in their Arbeite Hohe Else.

Maybe I should say that with marbles in my mouth, or something, to make it come out right.

MR. LASKIN: Very commendable.

Could I, coming back to table fourteen for just

THE WITNESS: (cont'd.) for the simple...of five point three relative to nine point one or one point one or one point three exists, so they are roughly in the same ballpark.

In terms of fiber size distribution, I think there are effects there as well, and some of the differences may be a result of that, because you...and this covers kind of two of the issues that you were talking about...processing versus as well. As you start in mining the effort is to try to keep fibers long because you get more money for the longer fibers. As you then separate out the dregs of the mill and put it into asbestos cement and use it there, or as you manipulate asbestos insulation material first in manufacturing and then in use, you are breaking fibers apart and workers are exposed to a correspondingly greater percentage of small fibers...the effects of which I think are not totally innocuous, and I've already dealt with that.

So that some of the differences that exist can be due to different percentages of the smaller fibers relative to the number that are measured.

I don't think there is much effect from other cofactors that are present in the particular processes that we are speaking of. The evidence for that is relatively limited.

The intensity of exposure is another issue that may have a, may play a role. I don't think it does so to an extent any greater than those that we've already discussed, which are variables that certainly play a role and lead to the variability. The possibility is that intense exposures may overwhelm clearance mechanisms and thus you have more fibers retained in such circumstances.

The other possibility is that with such intense exposures you end up generating fibrosis in localized areas, and that either prevents the transport of fibers across lung membranes and thus reduces risks of mesothelioma, and they...

THE WITNESS: (cont'd.) lower intensity exposures having correspondingly greater risks because you have unhindered mats of fibers that may have affect.

You can make arguments both ways. I think they are certainly...you know, whatever arguments one makes cannot be justified in terms of data. Some data are suggestive one way or another, and they do, I think, overall contribute to the variability, and I think that's about all one can say.

MR. LASKIN: Well, for example, one of the things that struck me was the relatively high risk factor in the amosite insulation workers, who, as I understand it, were, according to your work, exposed to very intense doses of asbestos, but over a relatively short period of time.

THE WITNESS: Yes, but they were exposed to a lot less asbestos than we found in the textile mill at Manville. There the concentrations were...by the company stated to be between fifty and a hundred fibers per millilitre...I mean, a whopping exposure...I mean, you couldn't see across the room, and reflected in the percentage of deaths from asbestosis in that group. Those people that were employed in the textile mill in our study of Manville, forty percent died of asbestosis.

Whereas from the Patterson group, the overall percentage of deaths from asbestosis is somewhere less than ten, around five percent, I believe. I remember it rising to something like fifteen percent in those that were employed for longer than two years, so we are talking about exposures both in terms of measured intensity and in terms of its effect as a manifestation of asbestosis considerably greater there, and yet the cancer risk in that group turned out to be the least of all the groups that we studied in Manville. Part of this is because, of course, they were dying of asbestosis before they reached an age old enough to have a high risk of malignancy.

But the surprising thing was that even in the

THE WITNESS: (cont'd.) lower exposure category the relative risk of cancer remained fairly constant, whereas that for asbestosis decreased. It would appear that the risk of cancer will decrease only at concentrations well below those where a significant risk of death from asbestosis is occurring.

When you look at factory circumstances, this really great intensity of exposures don't seem to have that much of an effect on mortality beyond a certain point.

In terms of the differences of methodology, looking at retirees clearly has a significant effect in that... just the curves I showed earlier where you have this rise and this fall, and you are looking only at the point of say forty-five years after first employment at age twenty, you've got perhaps one-half the risk that would have obtained earlier. You are looking at a survivor population that has significantly less risk than would be experienced in the same group of individuals exposed in the same circumstances, but studied during their working lifetime. There is considerable data that show that difference.

In fact, in Manville employees, where just looking at one plant one finds roughly ten times as much mesothelioma as Enterline had in his study in all plants considered, because the deaths were occurring before age sixty-five, before they entered the followup that he undertook.

I think what one has is a lot of these variabilities that we can't fully quantitate and make adjustments for. There is going to be many more in the different work circumstances that we are in fact having to deal with now and in the future, so when you are setting standards one has to protect everybody. You have to protect people that may be in these unusual circumstances that are contributing significantly to higher risks here, and the worst mistake that one can make is to choose as a guide the measure

THE WITNESS: (cont'd.) that is the lowest, that indicates the lowest risk, because that is protecting virtually no one. When you have evidence that much higher risks obtain in other circumstances and where you are dealing with those circumstances, one has to look to the distribution of risk that you would estimate from the variety of other exposures that could be seen. Overall, when one looks at the data on table fourteen, with all the caveats that you can put in...and there is no question that many uncertainties exist...you have, in eleven studies for which some dose-response estimates can be made, and when I was making them I was, I think, very conservative in fact on the conversion factors between particles and fibers. Enterline's, for example, would have had, I think, three is an overestimate there because much other dusty material was used in many processes that would have altered that value. One still comes to the finding that six of the eleven would indicate that there is at least a doubling of risk of lung cancer, and an increase of all asbestos disease, of ten percent for a lifetime exposure to two fibers per millilitre, and three of those studies would indicate that such disease would occur at lifetime exposure, forty-year exposures, to half a fiber per millilitre.

With so much evidence indicating such considerable disease at what are now currently existing standards, indicate that we have a long way to go before protecting workmen adequately in asbestos-exposed circumstances.

MR. LASKIN: Just so...and we'll break for lunch...but just so that I know the studies you are talking about, I take it the three that you are talking about that will produce a doubling of the risk of lung cancer at a half a fiber over a working lifetime, are the Patterson, New Jersey study of Seidman, the Dement study...

THE WITNESS: And the females in Newhouse and Berry.

MR. LASKIN: Q. (cont'd.) a moment, just ask you a few more questions? I note that two of the studies you refer to in table fourteen are, first of all, the McDonald, et al, study in Quebec, updated to 1980, and second, your own study of, I take it, a smaller cohort of miners from Thetford Mines in Quebec.

Can I ask you whether, notwithstanding the apparently different figures that appear for those two studies in table fourteen, whether in your opinion the results that McDonald produced were the same or relatively the same as the results that you produced?

THE WITNESS: A. Well, in terms of total attributable asbestos disease, there is a fivefold difference between the two. In terms of lung cancer it's about two and a half times on an estimated per-fiber basis. Some of this difference could be the methodologies of estimating fiber exposure, but...and the attribution of diseases to asbestos-caused, asbestosis, for example

The fact is that the two studies are... demonstrate lower relative risks either for lung cancer or for all asbestos disease than do other studies listed on that table. In that sense they are comparable.

Q. One other thing, as I understood it you looked at employees who had, I believe, been exposed for thirty years?

A. No, twenty.

Q. Twenty?

A. We used as our study group every employee that was a member of the work force and listed on the hourly seniority lists of four companies in 1961, so that all exposure categories within those companies were included. The large majority were from the Asbestos Corporation, what is now known as the Asbestos Corporation mine...gee, I forget the name...King Beaver.

A. (cont'd.) Additionally, they were all companies for which the work, in which the work force was organized by the Confederation Syndicat National....

MR. CASGRAIN: Syndicat National....

THE WITNESS: CSN. That is, there were two unions in Thetford, and we did this study using records that were provided to us of the company's seniority list by the CSN. so it was limited that way. But we included everybody on those lists.

MR. LASKIN: Q. Am I also correct that you were basically looking at long-term employees?

THE WITNESS: A. Right, everybody who had twenty years of seniority. I'm sorry.

Q. Is the figure that you used for McDonald, would his figure include not only long-term employees, but...

A. No, that included twenty-year...I selected the twenty-year people for that, if I recall.

Q. Only?

A. Twenty year and up, yes.

Maybe I should check, but my recollection was that was what I had done. I'm sure that's what I did.

Q. In other words, do I take it what you attempted to do was compare roughly similar populations as between yourself and McDonald?

A. That's correct.

Q. Apart from what you've already told me, that is perhaps ascertainment of cause of death, is there anything else that you can tell us that might account for the difference as between your results and McDonald's results?

A. The other factor was one that McDonald indicated in his papers, that individuals employed at Asbestos, Quebec, had a proportionally lower mortality and disease experience than individuals employed at Thetford, and this is

A. (cont'd.) within similar dust categories, and he, I don't think, had an explanation of that. Our data were at Thetford Mines, the higher of the two, the area which had the higher mortality experience.

Q. One other, one or two other questions on table fourteen.

Do I take it that the risk calculations in respect of the studies that were done out of Mount Sinai or the studies that were done by Seidman, et al, were based on national mortality figures in the States?

A. The study of the national cohort of insulation workers was based upon the national mortality rates. Let me just check what Seidman's was on.

Q. What about your own study in Manville, New Jersey, or Seidman's study in Patterson?

A. I'm looking at Seidman's at the moment. I know early on he had used national rates, but I think in the later periods when the better data were available he used the New Jersey rates.

Yes, he used the New Jersey rates, and that was...since he made the calculations I think that was also the case with the Manville study as well.

Q. From your knowledge...

A. In fact, I'm sure it was.

Q. ...is there a difference produced between using the New Jersey rates as opposed to using the national rates?

A. There is a little bit of difference. New Jersey is one of the leading states in the United States as far as cancer goes, and you go down the New Jersey turnpike and look at the counties along it and they are very high. In fact, they have been referred to as Cancer Alley.

The magnitude is about between five and ten percent.

Q. Higher than the national?

A. Higher than the national for certain sites. It varies around...for lung cancer, that would be the case.

I remember...the only data that I remember that I looked at recently enough to recall, is that of a study we just finished on printing pressmen in New York City, which has similar rates to New Jersey...that is, the city rates are similar... in which U.S. rates were six percent lower than New York City rates, and I think a corresponding number would apply to New Jersey, as I recall.

The differences between one set of data and another were extremely small, particularly compared to the mortality experience that is being seen.

Q. Another thing that struck me from table fourteen, and indeed has been commented upon before, relates... in some of our earlier testimony...relates to mesothelioma.

You noted in respect of McDonald's study in Quebec, the apparently low incidence of mesothelioma that was found. It would appear from even reading Dement's paper, where the relative risk in the South Carolina textile plant is high, nonetheless that there was a very small amount of mesothelioma.

Indeed, as I recall I think one death only out of the total deaths, and I'm wondering whether, at least in your view, that lends any support to the proposition that chrysotile...at least in respect to mesothelioma...is less hazardous than the amphiboles?

A. It's hard to say what the difference is and how much the fiber size distributions of the circumstances and the exposures contribute to the response. What one sees is a significant deficit in mesothelioma in mining circumstances, whether the mining be that of chrysotile or of amosite.

There is very little documentation of deaths from amosite in the South African data that are available, in

A. (cont'd.) contrast, for example, to crocidolite, with my previous statement applying to other than crocidolite. The difference between crocidolite and the chrysotile/amosite circumstances is that even in mining there, one has a very fine dust cloud with fibers readily fragmented into an aerosol with many of smaller sizes.

As one goes to circumstances where you manipulate the fibers and the exposure is proportionately greater in terms per fiber measured, one then sees mesothelioma in circumstances where pure amosite exposures are involved - the Patterson plant in particular - where amosite and chrysotile alone are involved - the insulation workers being the case.

With the insulation workers whose mesothelioma risk at equal times from onset of exposure considerably exceeds that of the Patterson workers at equal times from onset of exposure, one would have to attribute a contribution of chrysotile, as well as amosite, to that risk. There's something like, if I remember right, three or four percent of Patterson workers twenty or more years from onset of exposure have died of mesothelioma, whereas the corresponding number was seven to nine percent for the insulators.

Thus, I think to just kind of tie together the finding in France of numerous small fibers in the pleural tissue of individuals deceased with mesothelioma, and largely chrysotile fibers in particular because there are so many more small ones of them, and a much greater percentage of the smaller fibers there compared to the parenchyma would suggest a role for the smaller fibers, in part because of their greater number and their greater probability for penetration. So, I would not want to say that chrysotile per se is less likely to produce mesothelioma. I think one has to look at a lot more data than that, and I think the fiber size distributions are important.

If you, in fact, look at the animal circumstances,

A. (cont'd.) one finds that chrysotile is the most carcinogenic of all inhalation circumstances. So that the data that we would have is that in many of the circumstances that workers are currently exposed to, or most circumstances, and that excludes mining and it excludes textile production because few workers are employed in those industries relative to the number that are exposed to asbestos, that it would be imprudent to utilize what appears to be very unique experience in the mines and perhaps in the textile operations for consideration of a standard.

Q. Then do I take it that the conclusion that you draw from all of this is that we really can't say on the basis of the studies to date that one fiber type is more or less hazardous than another?

A. I would not attribute a major difference beyond exposure factors to fiber types.

Q. Would you attribute any difference whatsoever?

A. It is possible, but it is within the variability of the different dose-response relationships that we find. They are sufficiently uncertain that it's not possible with accuracy to say that something is two or three times more or less hazardous than another fiber.

Q. Is the other side of the coin, of that question, that whatever differences appear are, in your professional opinion, not statistically significant differences?

A. Well, we are not talking so much about statistical significance. I think there is a statistical significance between the lower rates in chrysotile mining and that in manufacturing and product use. What I am saying is that when you look at the ...I'm sorry...the circumstance significance is what I'm trying to say, there is enough uncertainty of a nonstatistical nature, of a perhaps real nature, in the studies that would preclude a definitive statement that

A. (cont'd.) one fiber per se is more toxic than another, more carcinogenic than another.

Q. You include, I take it, amosite, chrysotile and crocidolite when you make that statement?

A. Yes.

Q. I take it one of the things you have been saying though, is that you might find in any particular fiber count a greater percentage or a greater number of fibers less than five microns in length among the amphiboles than amongst the chrysotile fibers, for the same count?

A. No, there is a greater percentage of smaller fibers for the chrysotile than the amphiboles...than amosite. The percentage of smaller fibers for amosite is...the percentage of fibers shorter than five microns in an aerosol of amosite asbestos is less than the percentage of fibers smaller than five microns in a chrysotile aerosol.

Q. Is less?

A. Yes. What I'm saying is that there are enormous numbers of variabilities in all of these parameters that are virtually impossible to separately reconcile, and thus separately have a factor that you attribute to the size distribution, that you attribute to the fiber type, that you attribute to the exposure circumstances, that are unique to the different operations that are studied. To try to do so and to say firstly that there is a factor for chrysotile per se versus amosite per se, then to have a factor for size distribution of chrysotile and another factor for the size distribution of amosite, and another factor for the other things that you would add to it, would lead you down a morass of such conflicting and ambiguous data that you would have nothing at the end.

The data are sufficiently strong that in the circumstances of use and circumstances that we find ourselves

A. (cont'd.) trying to deal with exposures of populations to asbestos, that the obvious thing that should be done is to eliminate all exposures to the absolute extent that we can. I think the opportunity for doing so at this time is with us. We have limited amount of exposure in the factories, and engineering controls are developed that can reduce concentrations in most circumstances to extremely low levels.

The problems that we have in terms of control are those...and we'll come to this momentarily...are in dealing with all that asbestos that is out there in place, and there, I think, fiber numbers in aerosol measurements have fairly little relevance, because you are never going to have somebody around taking a measurement when the exposure takes place that will allow you to control it, and you are not going to control it in ninety-five percent of the current exposure circumstances. It's, control with fiber counting in a plant is easy. Control for fiber counting at a construction site is not, or in any building operation, and there one is...should operate on the principle that inhalation of asbestos carries with it a risk and to the extent that you can reduce that risk, you should. So that one need adopt largely procedural methods to devise ways that people required to undertake maintenance activities or removal activities, or otherwise in the course of their work deal with the asbestos that's there, do so in a safe way, both for themselves and for anybody else that might be involved. This largely is a matter of having proper clothing, proper respiratory protection and proper activities identified and utilized.

To try to put a factor of two between amosite and chrysotile is really missing the point, I think, and I'm kind of...very strongly feel that your doing so is dealing with trying to control something by...trying to control a sinking ship by bailing with a cup, when you have to get some buckets.

DR. UFFEN: Counsel, could I just...?

MR. LASKIN: Sure.

DR. UFFEN: I found a statement in here that I just want to check with you. It's on page twenty. It says,

"With respect to the studies of Enterline and Henderson in 1973..."

DR. DUPRE: This is tab nine?

DR. UFFEN: Yes, I'm in the blue book.

THE WITNESS: Yes.

DR. UFFEN: ..."The authors Enterline and Henderson at first suggested that crocidolite might have a higher carcinogenic potential than amosite or chrysotile. However, in the later analysis no such conclusion could be drawn from an analysis of the mortality rates of all individuals."

This is essentially what you have said. They drew the conclusion there...

THE WITNESS: They did not...no, they did not draw a...when they made an...what they did in a further study was to look at the relative mortality of those that had an exposure to crocidolite and who did not. On the basis of that, they did not draw the conclusion that crocidolite was...could not draw a conclusion that crocidolite was...

DR. UFFEN: Did they just not draw it, or did they...

THE WITNESS: Oh, no.

DR. UFFEN: ...withdraw the original?

THE WITNESS They didn't withdraw it, but let me read the exact statement, because I can find it. It's in this book, if I remember right.

DR. UFFEN: Okay.

MR. LASKIN: You are in the New York Annals book?

THE WITNESS: Yes.

MR. CASGRAIN: Dr. Uffen, which page are you quoting from?

DR. UFFEN: I read from page twenty, tab nine.

MR. CASGRAIN: Thank you.

DR. UFFEN: About the tenth line down.

MR. CASGRAIN: Thank you.

THE WITNESS: Well, in looking at the table of amosite only...of amosite or chrysotile only, they have SMR's that range from...for all individuals...from about two hundred to three hundred and sixty.

For the crocidolite exposure it goes from one hundred and forty-two to four hundred and sixty-one, and they found that there was a higher risk of cancer in the hundred and twelve men exposed to chrysotile and crocidolite in the...that were exposed to jointly chrysotile and crocidolite.

"This high SMR is clearly due to the asbestos-cement pipe workers, however. The sixty-eight men who had crocidolite exposure but who did not work in the asbestos-cement pipe manufacture considered of ENR workers who had only sporadic exposures, and of men employed in the production of asbestos gaskets and packing where crocidolite exposure was relatively low"... and that group had relatively less of an SMR.

Then they go on, 'because the number of individuals exposed to crocidolite who did not work in asbestos-cement pipe manufacture, it is difficult to draw a conclusion on the issue of whether the exposure to chrysotile, to crocidolite or not to crocidolite demonstrated an increased risk in their study."

MR. LASKIN: Q. Just a couple more questions

Q. (cont'd.) on this table.

Can I just come back to just one or two questions about those factors that I mentioned this morning?

A. Yes.

Q. We dealt with fiber type and I think we have dealt with fiber size distribution. Can I just summarize in that respect. When we talk about fiber size distributions, are we talking both about the breakdown of larger fibers into smaller fibers as you go through the processing practice, and the relative distribution between fibers greater than and less than five microns in length? Are we talking about both of those things?

A. Yes, well I think the breakdown of fibers as you go through the process will alter the distribution of fibers greater or lesser than five microns, so the two are intimately linked.

Q. All right. Is it...you'll have to help me.. is it the fact that the smaller the fiber, I take it, the easier it is in some respects to get into the lung and the pleura? Is that the theory?

A. I think there is more of an important...I think the smaller fibers...the differences...I think penetrability of body membranes is a more strong feature of small fibers than is the inspiration and deposition in the lung, because you are dealing with fibers, most of which are under one micron in diameter. For those fibers greater than one micron in diameter, you certainly do have a reduced respirability and deposition. But for those shorter than...I'm sorry...for those thinner than one micron, more or less irrespective of the length, the respirability is pretty much the same. This is from work of Timbrell. They are likely to be carried down into the...at least the terminal bronchials. But even there, there can be some deposition effects depending upon size, that would have the smaller ones be carried further

A. (cont'd.) down.

Of course if you get to the very, very tiny ones, they can be more easily expired as well.

Q. When we talk about...

A. So that the peak that's in the distribution is in terms of the deposition that would occur.

Q. When we talk about size, we are referring to diameter as opposed to length?

A. No, we are largely talking about...in all the discussion that went on with reference to fiber size...we were talking about length. The dynamics of deposition actually largely depend upon diameter. Large diameter fibers more readily impact, and virtually no fiber greater in diameter than three microns in diameter can reach the alveolar spaces.

The length is less important in terms of deposition. The fibers tend to line up along an air stream and be carried down with the axis parallel to the air stream, and not deposit on the various body membranes until fairly well down the lung tree.

Q. Okay.

A. Is that kind of the question you were asking? You seem uncertain.

Q. I suppose what I'm trying to get out in my own mind is, what is it - length, diameter or both - that have this health effect?

A. I don't think we really know what has the health effect, and that I would want to emphasize. The data on some overall health effects indicate that...again, when you use small aerosols...with inhalation, you find significant effects with a small diameter, even shorter length, the very super-fine chrysotile.

On the other hand, when you do deposition implantation studies or injection studies, now the data of

A. (cont'd.) Pott and Frederick and of Stanton would suggest that the longer fibers have importance, have more importance in producing cancer when they are at a site where the action can take place.

I said earlier that I think there is no question that once there, a longer fiber can indeed be more important. Firstly, it often can break up and produce smaller ones and you have twice the effect of a smaller one right away.

Q. It's got to get there in the first place.

A. There is a factor involved in it having to get there, and that tends to favor the smaller fiber. The size that is...and I discussed at least the inertial deposition in the lung tree, which is more related in diameter than in length, but I think in penetrability, length is important as well.

But overall, there is still number there. You have such a vast number of these shorter-than-five-micron ones that their effect cannot be dismissed.

Again, I go to the issue, if you can eliminate exposure to the ones longer than five microns, you would be eliminating exposure to the ones shorter than five microns as well, and what we should be doing is eliminating exposure to all of the fibers. That's a far better approach than to try to see if we can start altering various parameters and figure that this set of circumstances will be safe and that set of circumstances will be not, when the data that would support that conclusion is not there.

Q. The third matter was the possible involvement of a cocarcinogen, and as I understood your testimony before lunch you say there's just no evidence to support any of that kind of proposition?

A. I don't think that's a major factor. That is, other materials adsorbed onto asbestos that may be present in the product, or metals that can be present from milling

A. (cont'd.) operations, as have been suggested previously, would explain any of the data that we are seeing. I just don't believe that those sort of added factors are important. Certainly other factors, other exposures, particularly cigarette smoke, have a very dramatic effect.

Q. Sure. All right. We have dealt with the intensity-of-dose argument, and then the fifth one that I noted is whether the fiber itself undergoes any physical change, or whether there is some change in the integrity of the fiber that might make it more or less toxic. Is there any evidence of any of that kind of proposition?

A. The data are, again, somewhat equivocal here. You look at the implantation studies and find that the chemistry of the fiber plays very little role. That is, when Stanton put some aluminum oxide, fiberglass of different types, different asbestos varieties, onto the pleura of rats, he got...if the fibers were fine in diameter and largely longer than five microns, he found that they produced cancer with equal probability, roughly.

It appeared that the size and shape of the fiber, rather than the chemistry, were the dominant factors.

On the other hand, when you look at some experiments where you seriously alter the surface structure of the materials, biological effects can be changed, in particular hemolysis experiments do appear to depend upon the surface integrity of a fiber. Thus, some of the experimental work where severe physical alteration of fibers and reduced toxicity of smaller fibers that would be produced by such vigorous action might be explained in terms of alteration of surface properties, and the greater ease with which the fibers can be dissolved and altered by body fluids or otherwise.

The data that we are speaking of are either in in vivo experiments or just analysis, physical analysis of

A. (cont'd.) the materials themselves, and their action in fluids.

While they are suggestive of an effect in the body, it's not necessarily clear that will take place and how it will take place. Again, it's a sufficiently complicated process that it would be hard to make a definitive statement that these certain set of exact properties of a fiber are the ones that are producing cancer, and another set of properties are not.

Q. Fair enough.

I take it on the last matter which encompassed all of the methodological differences and so on, would it be fair to say that there have been methodological differences in respect of all of these various studies, but in and of themselves would not account for all of the difference in results that one sees?

A. I guess it's fair to say that we cannot account for all the differences in the results that we see. And we certainly don't understand fully the factors that lead to the differences. Whether we ever will is not likely, because we are dealing with exposure circumstances that took place thirty or forty years ago for many of these people, and reconstruction thereof is an extremely difficult thing.

Again, the issue is if we don't let any of this happen to individuals we won't have the disease.

Q. We won't have a problem.

A. I think that's the consideration that should be made paramount, rather than to emphasize total understanding of the process.

Q. I suppose I should ask you this one final question. Is there anything in table fourteen by way of accounting for the differences, that we haven't discussed?

A. No, I don't think so.

I would just like to read actually the first

A. (cont'd.) paragraph of this paper, which everyone can get later because it's not available right now, because it kind of has a very good quotation that was made and that bears right upon the issue that we are talking about, that was written in 1761, by a British physician, John Hill. He was writing in a treatise, the title of which is, Cautions Against the Immoderate use of Snuff, Founded on the Known Quantities of the Tobacco Plant and the Effects it Must Produce when This Way Taken into the Body, and Enforced by Instances of Persons Who Have Perished Miserably of Diseases Occasioned, or Rendered Incurable by its Use.

That's the title.

In any case, there are three sentences that are worth considering. This is now directly quoting Dr. Hill:

> "Whether or not the tumors which occur in snuff takers are absolutely caused by that custom, or whether the principals of the disorder were there before and snuff only irritated the parts and hastened the mischief, I shall not pretend to determine.
>
> Even supposing the latter only to be the case, the damage is certainly more than the indulgence is worth.
>
> No man should venture upon snuff who is not sure that he is not liable to cancer, and no man can be sure of that."

I think we can nearly substitute asbestos for the same thing.

In this statement Hill firstly focussed upon one important question of carcinogenesis, that of a multifactorial interaction, and we've talked briefly about that.

But secondly, he raised the additional question

A. (cont'd.) of threshold. He knew of none for snuff, and we have no evidence for asbestos.

Thirdly, of considerable practical importance, he pointed out that one need not know all the answers to the scientific questions in order to initiate control activities.

Finally, while lacking numerical verisimilitude, his cost/benefit analysis is as relevant as most that exists today.

Q. All right. I take it another of the matters, Dr. Nicholson, you would like to discuss with us today, is environmental exposures. Unless you have any further comments on the occupational side, perhaps we could turn to the environmental side.

A. Okay. I guess we could go to the slides.

Firstly, as one knows, the initial awareness of environmental exposures began with the finding of mesotheliomas in the Northwest Cape Province, by Wagner, who identified contact only with asbestos in individuals who were exposed only by virtue of living in that area, and then later the study of Newhouse on the background of individuals deceased of mesothelioma in a London hospital showed both neighborhood and family contact exposures to be of consequence.

Proceeding beyond that, and you will hear more from Dr. Anderson on this, but I just would briefly show his data on the...which is in the next slide...which shows that residents in the household of an individual who worked with asbestos, in particular those individuals at the Patterson plant, is sufficient to produce x-ray evidence of that exposure. The evidence is largely in pleural changes rather than parenchymal changes, and the extent is such that the individuals are not disabled, but the exposure was sufficient to produce such abnormalities in approximately thirty-five percent of family members of former workers, and it didn't matter too much what the relationship was. The rough value of thirty-five percent obtains for all groups.

A. (cont'd.) This exposure has clearly been shown to be sufficient to produce disease....in particular, mesothelioma.

Just for comparison, the next slide shows the number of mesothelioma cases in the nine hundred and thirty-three factory workers that provided the contact with asbestos. There fourteen deaths have occurred, and these data are...I guess you want it identified...in a paper by Anderson, presumably published in the same Annals.

If I had known I would have to identify everything, I would have taken a lot of those slides out.

MR. LASKIN: I'll find it.

MR. CASGRAIN: Is this amosite exposure?

THE WITNESS: Yes, this is amosite exposure. That's correct. It's the Patterson plant.

MR. LASKIN: I think it's...yes, I believe it's the paper, Asbestosis Amongst Household Contacts of Asbestos Factory Workers.

THE WITNESS: Yes. What's the page number?

MR. LASKIN: Beginning at page 387.

THE WITNESS: Okay.

Yes, the x-ray data are provided in far more detail, as a matter of fact, in table six and seven, and ...

MR. LASKIN: Q. Can we just go back to the preceeding slide so we can identify it?

THE WITNESS: A. Yes. Much more detailed data than appear there are in tables four through eleven of the paper by Anderson. In fact, the slide largely is a duplicate of table twelve, which has fifty-six more...fifty-three more cases included. This was a slide made of the group prior to the followup that Anderson reported on.

MR. LASKIN: Just to help everyone, Dr. Nicholson, the paper, the statistic, I think, comes from a paper

MR. LASKIN: (contd.) by Anderson et al, and will be before this Commission when Dr. Anderson testifies next week.

THE WITNESS: Oh, in the text of that paper, on page 398, he discusses the finding of four mesotheliomas among this group of workers, and I'll show you in the next slide the detailed breakdown. I'm sorry...the slide after that has that information, according to time from onset of exposure.

But first let me go to the previous slide which is useful for comparative purposes and represents the deaths from mesothelioma in the working group, and these data are available in the paper of Seidman et al, which begins on page 61. Not as that table, but they are available in that form.

Where fourteen deaths occurred, all after twenty years from first exposure, and these represented approximately two percent of the total deaths that occurred between twenty and thirty-five years from onset of exposure... I'm sorry...not two percent, nearly four to five percent. That is, fourteen out of three hundred and four.

The next slide shows the corresponding data for the family contacts, in which four deaths, all after thirty years from first exposure, occurred among three hundred and eighty-four deaths of the family members after twenty years from first exposure. There were two thousand, two hundred and seventy-two identified family contacts that provided the population among whom these deaths were observed, so we are talking here about one percent of deaths after twenty years from first exposure, and approximately two percent of those subsequent to thirty years from first exposure. Whether the later observation will lead, as expected because of the high increasing risk of mesothelioma with time from onset of exposure to a greater percentage, will only be seen with further followup over the next ten to fifteen years, of this group.

THE WITNESS: (cont'd.) But this is a considerably important contribution to mortality, and a current study underway now of lung cancer among these same family contacts indicates that lung cancer, too, will be elevated among this group of two thousand, two hundred and seventy-two individuals.

The knowledge of the exposure these people had that led to their disease is not known. There, as well as in the other environmental circumstances, we cannot quantitate the dose that produced the risk, although it makes some comments on possible contributions in family...various possible contributions of asbestos exposure in environmental circumstances, but these are data that are measured in recent years with what were fairly severe circumstances of severe environmental contamination, and they cannot, with certainty, be equated with those that took place that produced the disease that one sees.

But whatever the levels are that are required to protect individuals against risk from asbestos disease, they clearly have to be significantly less than those that obtained in households of workers.

The next slide.

MR. LASKIN: Q. Just before we get there, do we make anything of the fact that the mesotheliomas in the household contact situation appear to be occurring later?

THE WITNESS: A. I think that's the same issue as we have discussed earlier, the risk is lower, and so the curve that is rising rapidly with time is a curve that is lower than that which would obtain for the workers, and thus the difference in the risk at a given point in time, which would reflect that dose leads to a probability of significance occurring somewhat later in time with the family contacts.

Also, we are dealing, clearly, with very small numbers, and the happenstance of, for example, not having one

A. (cont'd.) case in twenty-five to twenty-nine years is probably just that, perhaps. I don't think we can make anything specific out of it. I think that where we have data of substance on the time course, the statements that I made earlier apply, and the numbers here do not contradict them.

The next slide.

MR. CASGRAIN: For the purpose of eventual cross, is this being identified in any way, shape or form as a tab?

THE WITNESS: Not as a table. Oh, that table just provides the breakdown by years from onset of exposure of four cases that are discussed in Anderson's paper. It does not...it appears in my...it must appear in this thing, I'm sure. It's in tab nine, exhibit nineteen. It's table nineteen.

I'm sorry, that's the previous slide. It's... both the previous slide and this slide are in table nineteen.

DR. DUPRE: Of tab nine?

THE WITNESS: Of tab nine.

DR. DUPRE: Yes, I see it there.

MR. LASKIN: Thank you.

DR. DUPRE: Do you see it there, Mr. Casgrain?

MR. CASGRAIN: Yes, I have it before my eyes now.

THE WITNESS: So it's there.

MR. CASGRAIN: Now I have it.

THE WITNESS: Okay. Next slide.

I guess one of the extreme circumstances of family contact exposure was brought to light a couple of years ago when a woman with mesothelioma came to Mount Sinai after having been seen elsewhere. This is her x-ray in 1977, which was completely normal. In 1978, she developed...the next slide... developed mesothelioma and later was diagnosed on the right side... on the left side of that slide, on her right side.

A. (cont'd.) She, after feeling pain in her chest, went immediately to her doctor because the symptoms that she experienced were identical to those of her mother, who had developed mesothelioma a few years earlier. Each were exposed in the household. Her father was an insulation worker at the Quincey Shipyard, and the mother shook his clothes out on the porch steps and received her exposure in doing so. The child was playing nearby when this occurred, different times, and thus received her exposure. Each died of mesothelioma. The father died of lung cancer. So the entire family was affected by the one person's occupational exposure.

The next slide, please.

Next slide then. Okay.

The other circumstance of importance for asbestos disease is what has been called by several people, bystander disease. It's really not bystander disease, it's byworkers, because the people were actively engaged in their trade. It was first identified as a major problem in shipyards by Harries, in 1968, and here are data of Harries' in the reference provided there, the Environmental Research, Volume 11, which documents fifty-five cases of mesothelioma among employees of the Davenport Dockyard, only two of which occurred in individuals whose trade required the direct use of asbestos on a regular basis. The others were shipwrights, boilermakers, engine fitters, electricians, and their opportunity for exposure came from some occasional work with asbestos, but largely from the exposure that the insulators or the spray personnel provided in the course of their work.

You can see the problem illustrated in the next slide, of where an insulation worker now, fortunately, at least wearing somewhat of a mask, is mixing asbestos cement, and this is where high concentrations of asbestos certainly obtained. The pipe fitter in the background in this power plant

A. (cont'd.) slide is not, and he didn't know what the consequences were of his work activity there.

The next slide, when this sort of problem takes place in the...oh, dear...hold of a ship, with the very confined spaces in many trades employed, the exposures can be fairly significant.

The next slide, please.

This shows some of the data that we have recently obtained of the prevalence of x-ray findings among long-term shipyard employees in a yard in which repair work was done, dating back well before World War II...the Key Highway Shipyard in Baltimore, Maryland. These data are published in an article in the American Journal of Industrial Medicine, the December, 1980, issue, and I have supplied copies here which can be available, but the substance of the data are that overall roughly eighty-five percent of those individuals with twenty or more years of exposure have some x-ray abnormality characteristic of asbestos exposure. Only thirty-nine of two hundred and eighty-two individuals have normal x-rays. A considerable amount of abnormalities consisted of pleural changes. The intensity, the degree of the changes is, fortunately, less than those seen in insulation workers where at the same times from onset of exposure there are a greater percentage of category two and three abnormalities than are seen here. But even so, the relative disease present in this group of individuals certainly raises concern.

DR. MUSTARD: Can I ask a question?

THE WITNESS: Yes.

DR. MUSTARD: Of your total abnormal, you've got twenty-five out of thirty-seven for the twenty...

THE WITNESS: Yes, and that's not thirteen point one. That's a misprint. I think it's seventy-three percent of whatever...the twenty-five over thirty-seven is the

THE WITNESS: (cont'd.) correct value. The percentage is wrong.

DR. DUPRE: Can we pause for a moment for the record, while my learned counsel instructs us all as to whether this is a tab number or what?

MR. LASKIN: I haven't got Mr. Warren here to help me, but let's mark this article entitled Radiological Evidence of Asbestos Disease Among Ship Repair Workers, reported in the American Journal of Industrial Medicine by Selikoff, Dr. Nicholson and Ruth Lilis, as tab twelve of exhibit nineteen, so that we keep all of Dr. Nicholson's publications in one place.

DR. DUPRE: Thank you.

EXHIBIT # 19, TAB 12: The abovementioned document was then produced and marked.

THE WITNESS: The only other thing I would mention about the study is that in the course of the examination of the two hundred and eighty-two individuals, we identified five with lung cancer that had no previous knowledge of disease, and this is a remarkably high finding and points not only to the concern of fibrotic disease, but for malignancy as well.

The issue overall of the effect of past shipyard work in the United States is of obvious concern to HEW and many in the United States, with much publicity being generated about such exposures, considering that four and a half million individuals were employed at one time or another in wartime shipyard work. Estimates can be made of the possible asbestos exposure from such employment. Actually there is an equal, if not approximately equal amount of disease that will be generated in the continuing employment in shipyards subsequent to World War II, because there the individuals had much longer employment. While there was a great many more in

THE WITNESS: (cont'd.) World War I, the total employment time was relatively short, the average employment being about one year. So in essence, what you are talking about in World War...I'm sorry, World War II...World War II, is four and a half million person years of exposure, whereas subsequent to that an average work force of two hundred thousand was employed for thirty years, and that generates six million person years of exposure, more than that of the wartime group. As these individuals are, the greater percentage of these individuals are alive, our experience in the United States in future years will have a greater role...there will be a greater role played by that employment than by the wartime exposure.

The next slide.

This illustrates what is in fact likely to be an even greater problem than the exposure in shipyards, again both because of the significance of the exposure and its intensity, and the number of people that had this exposure, and that's the use of asbestos in construction, particularly for the fireproofing of high-rise buildings, and it's certainly a problem that exists in Canada.

Here you see a picture taken in the early 1970's of the spraying of a building in the Wall Street area, with no effort to confine the spray to the job site. This is one of the better jobs where at least ten or twenty percent of the material is landing on the steel, most of it, of course, going elsewhere.

The next slide shows the overspray when contained within the site and after the floor is done, the standard procedure at that time was simply to lift the curtains and let it blow away after, perhaps, some effort to sweep a little bit of it up. But again, there was very little concern to localize contamination to the job site itself.

The next slide shows some of the exposures

THE WITNESS: (cont'd.) that occurred on the work site, to the bricklayer...the next slide...the electrician... next slide...and to sheet metal workers who will be hanging ducts underneath the applied asbestos material. As they put their hangers up, they knock the material down and all subsequent activity in those circumstances leads to exposure of the worker directly involved, and to others as they re-entrain the asbestos into the air of the building.

The next slide shows...I don't want to show it, then you'll ask where...let's leave that slide out.

The fact that many exposures can take place is the main point I'm trying to illustrate here.

With the number, with roughly three to three and a half million workers employed in construction trades at any one time in the United States, and with high rise construction employing a very large portion of these people, you have a vast potential in years to come of disease from that exposure that would have taken place in those occupational circumstances. And it, in fact, is likely to exceed the contribution of asbestos disease in the next two decades from shipbuilding.

The legacy of that procedure is also manifest in the finding that one can measure excess chrysotile air levels near spray fireproofing sites that go to as great as three hundred and seventy-five nanograms per cubic meter of air, where the quantitation of asbestos is utilizing chrysotile, which can be identified in an electron microscope by its tubular structure. Additionally in some of the exposure circumstances, an exposure to amosite would occur as well. Because of the much greater analytical difficulties of identifying each individual amosite fiber, we were using chrysotile as the measure of environmental contamination in this and a variety of other studies that I will now describe.

MR. LASKIN: Q. Could I just stop you for a

Q. (cont'd.) moment, just so we can get our measurements straight?

A. Yes.

Q. A nanogram is a weight measure?

A. Right.

Q. Somewhere I read...and you correct me if I'm wrong, I think I read it in your material...that a thousand nanograms roughly corresponds, if you did a fiber conversion, to point zero three fibers per cubic centimeter?

A. That's correct. The data that we, as well as others, have would suggest that that conversion is appropriate. The uncertainty, and it is somewhat large, it might be a factor of two or three either way, so we are talking about clearly an approximate conversion factor. Part of this is because the percentage of fibers counted by optical microscopy is variable. As we have already seen in part, it's because there is also uncertainties in the accuracy of the measurement with the single electron microscope level. There's a factor of two uncertainty there, so that we are talking about a rough measure, but that's the approximate conversion factor that would be applicable.

Q. Just one other measurement factor so we have it in perspective. As I understand it from the city sampling that appears throughout your articles, generally speaking if there is no asbestos exposure...

A. That's the next slide.

Q. ...the background measurements will be around, or less than fifty nanograms...?

A. That is correct.

Q. Per cubic meter?

A. Yes. The next slide shows those data.

MR. CASGRAIN: You asked a question, I didn't get the answer. You asked what the background was?

THE WITNESS: Yes, and I'm now giving you the answer.

MR. LASKIN: He's giving us the answer, Mr. casgrain.

THE WITNESS: This is why I moved on, because this is exactly what he was asking about.

For comparison purposes, the concentrations that ranged from roughly nine nanograms per cubic meter of air to three hundred and seventy-five in the previous slide, about sites of spray asbestos, can be compared to those that we have measured in twenty-four...in actually composites of twenty-four hour samples taken in forty-eight United States cities. This is a table of the cumulative distribution of samples with a concentration less than the specific level.

For example, take the line Asbestos Concentration Less Than Ten Nanograms Per Cubic Meter of Air. There were one hundred and seventy-six samples that had a concentration... one hundred and seventy-six out of one hundred and eighty-seven that had a concentration less than ten nanograms per cubic meter of air. In other words, ninety-five percent of the samples had a lower concentration than that. Ninety-eight percent had a lower concentration than twenty.

We had one sample, I think two samples exceeded fifty nanograms. One was about fifty or sixty. That was in a city that had a very large shipyard.

The other, which was ninety nanograms per cubic meter of air, was in a city that had four uncontrolled brake lining manufacturing facilities operating at that time, as we've found out subsequent to this analysis.

DR. DUPRE: Excuse me, Dr. Nicholson, is this slide the same as tab six, table four?

THE WITNESS: Actually, every slide on air concentrations will be found in a tab of Selikoff's. One of my papers got put there.

DR. DUPRE: I think that I found it in table

DR. DUPRE: (cont'd.) four of tab six, which is your...Nicholson et al, Control of Sprayed Asbestos in School Buildings?

THE WITNESS: Yes, that's right.

DR. DUPRE: On table four...

THE WITNESS: No, that's not...yeah, it's on table four.

DR. DUPRE: ...I see numbers in the first two columns.

THE WITNESS: Well, let me also just refer to a paper where these different numbers are summarized. It's Environmental Asbestos Concentrations in the United States, by myself, Rowe, Weissman and Selikoff. It appears in his...do you have his?

MR. CASGRAIN: I will distribute it.

THE WITNESS: Well, you can distribute it right now, which will simplify matters. But it's already in your records.

MR. LASKIN: Could I, while we are distributing it, is a nanogram one-billionth of a gram?

THE WITNESS: A nanogram is one-billionth of a gram, and we are talking about a very small number of...a very small...

MR. CASGRAIN: One-billionth?

THE WITNESS: One-billionth of a gram.

MR. LASKIN: To the minus ninth power?

THE WITNESS: Right. That's, I think, what has been shown...that's right. to the minus ninth power.

The conversion factor between fibers and nanograms, that was mentioned previously, would suggest that one nanogram is the equivalent of roughly thirty to thirty-five fibers longer than five microns, plus all the smaller ones that would be in the average aerosol, which would be a hundred

THE WITNESS: (cont'd.) times as many, so we are talking about one nanogram probably corresponding to thirty thousand fibers...I'm sorry, three thousand fibers in a typical chrysotile aerosol. That is the conversion that was discussed previously, one thousand nanograms equals point zero three fibers per millilitre, leads to the corresponding conversion of one nanogram, with no volume unit, one nanogram being equal to thirty to thirty-five fibers longer than five microns, plus their associated sub-five micron fibers, as measured in aerosols.

MR. LASKIN: Can we, just stopping for the record, just mark this latest paper, Environmental Asbestos Concentrations in the United States, as tab thirteen of exhibit nineteen.

EXHIBIT # 19, TAB 13: The abovementioned document was then produced and marked.

MR. LASKIN: Sorry, Dr. Nicholson.

THE WITNESS: The next slide bears upon kind of the problem that now continues with the past use of asbestos for fireproofing, because as buildings continue to be used, there is the need to install other equipment in this space, and as it is done, the asbestos that is there in place is knocked down and may recirculate through the building. Currently the World Trade Center, which is one of the buildings that has a large quantity of asbestos sprayed on it, is undertaking to install sprinklers in the entire twin-tower structure because of the concern for fires that has developed in New York. As they do so, the hope is that it be done without significant contamination.

As phone installers, electricians, plumbers, or whatever, undertake activities here, the dislodgement of asbestos is a real problem. It can be seen in virtually any building, in the next slide, by simply lifting up...I'm sorry.

THE WITNESS: (cont'd.) No, no, that's all right. I forgot about this slide.

The concern with such dislodgement is that the air supply system of the buildings is designed such that air is sent from a central fan room through ducts into a room, as would here, I guess, come maybe through some of the slots in the lighting fixtures, and then leave the room through perhaps, in this case, slots in other lighting fixtures. In general, office buildings do not have ducts into which the return air is directed. But it simply goes into the space formed by the hung ceiling, as in this room, and the underside of the floor above which, in many buildings, is sprayed with asbestos. Then it's carried across the building to a return air duct to be recirculated through the building.

When dislodgement of asbestos or erosion from air currents, or just loss of efficacy of binder occurs with the fiber falling down, fibers can be entrained in the building and carried throughout it.

The next slide...

MR. LASKIN: That slide, by the way, is page six of tab four.

THE WITNESS: ...shows what is a very typical finding in any building where this has taken place. You lift up the ceiling panels and you see the asbestos lying down on the top of the hung ceiling, either dislodged by maintenance activities or falling of its own accord. Some of the spray jobs were really atrocious, and there was no effort to tamp the material onto the steel work, and as a result it is highly friable and easily dislodged.

The next slide shows graphically the findings of the air concentrations that we have measured in a random sample of buildings in which spray fireproofing was utilized. This was...and it appears the entire study is...

MR. LASKIN: I think this is page twenty-seven of tab four, if I'm not mistaken.

THE WITNESS: Okay. It's tab four, in any case.

This graph is part of tab four. Tab four is a composite of two sets of data, but it is in tab four.

Here it just simply displays a percentage of samples that had a given concentration of asbestos in various concentrations, ranging from less than two nanograms per cubic meter of air, two to five, five to twenty, twenty to fifty, fifty to two hundred and above two hundred.

The data of the crosshatched are those data that were taken in buildings in which friable spray asbestos was utilized, compared to outside samples taken at the same time. In all of the outside samples, the air concentration was less than twenty nanograms per cubic meter of air. Half of the air samples taken in the buildings exceeded twenty nanograms per cubic meter of air, with one of the concentrations going up to as high as eight hundred nanograms.

So there is evidence of some asbestos contamination in a large number of samples in buildings that were not selected for the possibility of damage. We were just... the city co-operated with this structure and some guy went around and said, can we sample our building. Whoever let us do so, we did. It was a study done with the conditions of the buildings unknown to us at the time the sampling was done, and thus is likely to be typical of most high-rise buildings.

The data in the next slide show the detail of breakdown according to the same display as was given before for the outside samples. There, in contrast to having ninety-eight percent of the twenty-four hour samples less than twenty nanograms, here one finds fifty-four percent of the samples exceeding twenty nanograms per cubic meter of air. That is, looking at the twenty nanogram line, forty-six, forty-seven

THE WITNESS: (cont'd.) percent of the samples had a concentration of twenty nanograms or less.

The next slide...it's kind of a poor slide without darkness...illustrates another use of...it's a special concern, of course, of asbestos fireproofing, and that was its use in public buildings as acoustical soundproofing. Here is a building in New Jersey that was one of the worst, in fact the worst that I've ever seen, where the asbestos had been sprayed on a hallway that was relatively low...it's barely a foot or so above a doorway...and kids could easily reach up and disturb it and it was easily broken apart.

The air concentrations when the active disturbance takes place, as measured by us simulating damage in a building that asbestos was to be removed in, ranged from two to ten fibers per millilitre over a short period of time, so you could have the equivalent of short-term occupational exposures in a school situation.

The next slide shows, the next couple of slides, just show...that's kind of upside down...but shows the damage again. Now, in a less friable form of asbestos, a vermiculite asbestos matrix that was again damaged by kids...the next slide...oh, let's go back...shows again some severe damage in the first school at another location where virtually all of the surface has been affected.

We undertook a study which is tab six, of asbestos in schools in New Jersey, and investigated the condition in a sample of about fifty schools that had reported to the State that they had asbestos in pupil-use areas. This was fifty of a total number of approximately two hundred and fifty that had reported that condition to the State, the two hundred and fifty representing about twelve percent of the schools of New Jersey. The schools that we selected were done so on a random basis, to some extent, although the schools with the

THE WITNESS: (cont'd.) greatest area reported were preferentially visited. But the condition of the schools at the time of visit was unknown to us.

Of those visited, sixty percent of the schools had some damage in some areas, to the asbestos in the pupil-use area, and about twelve percent had relatively serious damage... serious being even less than this, but of a sufficient amount that there would clearly have been fairly significant exposures to pupils at the time that it occurred.

We undertook sampling in some of these schools, and the schools that were selected were those that did have damage, and thus it was not just a random sample as the previous one.

The sampling was done, though, at a time when no damage was taking place. That is, we investigated the air concentrations over a school day and monitored the sample taking to ensure that no disturbance of the asbestos occurred during the sampling.

The next slide, I think, shows the results of that analysis, where there was considerable evidence of contamination here, eighty percent of the samples taken...or seventy-eight percent of the samples taken had air concentrations in excess of fifty nanograms per cubic meter of air, with one being approximately two thousand nanograms per cubic meter of air.

This has led...findings such as this have led to major efforts in the United States, and also in Canada, I understand, to remove or otherwise preclude the dissemination of asbestos in public schools, because the concern for mesothelioma in years hence, albeit from exposures that are much less than those of the workplace...for example, the highest exposure here would be something like point zero five fibers per millilitre... but when you consider that you are exposing children as young as six years old, and that the population that are attending schools

THE WITNESS: (cont'd.) in schools with asbestos in place, total, I think, in the United States, six million pupils, the public health problem is a real one and thus the concern that has been generated, I think, is appropriate.

The next slide shows finally the last set of data that we have obtained in environmental circumstances, and that is in the homes of thirteen mine and mill employees, one set being in California and another in Baie Verte, Newfoundland, where the workers had no opportunity for showers or changing clothes at the job site and the homes that were sampled had the dirty overalls in the laundry area and you could see visible asbestos in the living areas of the home. The concentrations ranged from something, from about fifty up to a thousand, generally, but with one single sample of approximately five thousand nanograms per cubic meter of air.

The relationship of these concentrations to those in other circumstances in the homes where workers' families have developed asbestos disease is uncertain. We don't know whether such concentrations are typical.

But in a three or four year period of time, we have made major efforts to sample those environmental circumstances where the worst of conditions obtain...the circumstance where there is somebody on a twentieth floor of a building blowing asbestos out into the community, homes with the asbestos clothes and the fiber clearly visible on their floor, schools such as you saw that picture of - that school was part of the sampling program, and asbestos buildings where you had the material coming down. In eighty-nine such environmental samples, roughly half...forty-three exceeded fifty nanograms per cubic meter of air. But only two of the eighty-nine exceeded one thousand nanograms per cubic meter of air, even though we were looking very...we would have sampled any environmental circumstance of major concern.

THE WITNESS: (cont'd.) In contrast of all the ambient air samples that we have analyzed that were taken in other than circumstances of contamination, only three exceeded fifty nanograms per cubic meter of air. Thus, the finding of air concentrations between fifty nanograms and one thousand nanograms per cubic meter of air, I think are indicative of the potential for serious asbestos disease, and thus appropriate action should be taken to eliminate the possible exposure that could lead to such air concentrations.

DR. UFFEN: Could I just...that's a very significant statement there, and I want to make sure I understand it.

A few minutes ago you pointed out that fifty nanograms was the order...

THE WITNESS: You rarely see an environmental contamination in excess of fifty nanograms per cubic meter of air without an exposure circumstance.

UNIDENTIFIED SPEAKER: Fifty or fifteen?

THE WITNESS: Fifty - five zero.

DR. UFFEN: Now if the kids are exposed to that for fifteen and twenty years in a school, would their accumulated exposure at this admittedly small level get them up into the level of dose where the epidemiological data is pertinent for workers?

THE WITNESS: No, you are still at a...what you are is that the risk for an individual from such exposure is so minimal, is really extremely minimal. When you are talking about exposures, say, let's take three hundred nanograms per cubic meter of air, which would correspond to point zero one fibers per millilitre, we are talking about for...I really should do this separately for mesothelioma alone, which I haven't done, but let me just say that this is very rough. In

THE WITNESS: (cont'd.) fact I'll do it and when I come back tomorrow I'll give the results.

But you are talking about a risk of excess mortality for workers for mesothelioma somewhere around two or three percent, probably, at one fiber per millilitre...that is, for insulation workers we are talking about their exposure being around ten, and seven percent die of mesothelioma, so that maybe one percent is the risk of...one to two percent if you follow the insulators up for a lifetime...at one fiber per millilitre. This now is one one-hundredth of that exposure, but you get a much more significant factor, perhaps a factor of ten, because the exposure started much earlier in life.

You recall the conversation we had about the importance of early exposures in mesothelioma.

But whether it's one one-hundredth or one-tenth or even one one-thousandth that of a worker, when you now have this enormous number of children exposed, the consequences could be serious.

DR. UFFEN: I was trying to relate this to what you told us about when you first started out this morning, and whether we could take a slope from the graph and apply it to the kids.

THE WITNESS: The best information that we have is that that curve should be applied to the kids. That is extrapolating it downwards, but I would emphasize that we don't have the data at the lower end of that curve. What we have is a curve that is, over the region that one has data, has a power log dependence of the order of T to the fourth or fifth, with the likelihood that the position on a risk/age graph would depend upon dose...that is, and be linear related...that is, one-tenth the dose would have a curve down by order of magnitude but still have the same time dependence, and while the data for mesothelioma are less certain than the linear dose-

THE WITNESS: (cont'd.) response relationship obtains, that is the best data that we have and no data conflict with that, we should utilize that concept for control strategies in these circumstances. There are no better data that could be utilized.

MR. LASKIN: Just so that I'm clear, what model is it that you are suggesting we extrapolate from?

THE WITNESS: The time dependence of mesothelioma as seen in insulation workers. The dose...a dose dependence that would say that the risk for the given exposure at a given point in time is proportional to the dose of asbestos inhaled. So, a linear dose-response relationship and a time course given by the time course in insulation workers.

MR. LASKIN: Do you choose insulation workers because that's, in your view, where the most complete study has been done?

THE WITNESS: Yes. That's the only study that allows one to obtain the time course. There, there were a hundred and seventy-five deaths that allowed an analysis to be done, and the time parameter that is of importance is the time from onset of exposure, rather than the age.

MR. LASKIN: Fair enough.

DR. DUPRE: Can I ask the source of table six?

MR. LASKIN: I've seen that.

THE WITNESS: It's in this...what did you call it...tab thirteen?

MR. LASKIN: Yes.

THE WITNESS: Tab thirteen. I hope it's there. Yes, it's there.

DR. DUPRE: Table two?

THE WITNESS: Yes, table two.

I should emphasize that all of the samples

THE WITNESS: (cont'd.) that were shown in these last slides were analyzed in the same way. There can be variability in the results obtained by individuals who utilize different analytical techniques, and I don't want to get into the issue of what's right and what's wrong, but what we see here are samples in which there is comparability of analytical techniques, and so the evidence of contamination is thus, I think, established.

Go to the next slide. I think I'm nearly done.

Oh, yeah, I would just like to quickly illustrate the general problems where we now have...the problems that we now face with asbestos. It's not so much in the factory circumstances, particularly Ontario where one of your major factories has closed down. It's very...even at its greatest operation, mining has a relatively small number of individuals exposed compared to the number that are exposed subsequently. For example, what is there...in Canada, three thousand, four thousand, five thousand employed at one time in mining. Maybe alive today...I don't want to say because I'm only guessing...but of the order of tens of thousands at most, would have had an exposure in all mining operations in Canada and the United States.

In the United States, when one looks at...alone, when looks at the different trades that can have some exposure to asbestos, the number of individuals that would have exposure, either in maintenance activity, in shipyards, in construction, in primary and secondary manufacturing, in automobile repair, total something of the order of thirteen to fifteen million people. For many of these, the exposure is very minimal and the disease that will result therefrom would be also relatively limited. But it is in the end uses of asbestos that the number of people with exposure reached significance, and in some of the circumstances the intensity of the exposure is also

THE WITNESS: (cont'd.) considerable.

Let me at this point now, that is where we have to focus our attention.

The next slide, I think, illustrates in a bizarre way the point and the difficulty at times of monitoring many of these maintenance situations, which largely occur in various buildings.

The next slide...oh, I don't know how...oh, this was a steam tunnel in which a pipe was being removed, and insulation was taken from that pipe before the steel was taken out. The insulation material was put in an open cart, moved through this building leaving a trail of asbestos as seen here.

The next slide...we're nearly done with the slides...shows its transfer from the small cart, which is in the foreground, to a very large bin.

The next slide shows a truck which was the receptacle in another transfer from the large bin, and that truck was then driven through the streets of New York.

The next slide shows the location of the bin where all this transferring is being done, located directly underneath the air intake for the building, and if you go into the doorwar where all the asbestos is on the floor and read the sign at the first doorway inside, it said, Inhalation Therapy Unit, Mount Sinai Hospital.

So that happened in our institution in 1969, and it got that far before the activity was halted. This is still going on, things like this are still going on today in many areas, and it's the control of the one million tons of friable asbestos that is in place in buildings, in ships, on power...heating equipment and power plants, chemical plants, in the United States and Canada that are going to provide the exposures in the future, and the control measures that are to be adopted should focus on the particular difficulties of that

THE WITNESS: (cont'd.) control. Here, as I mentioned earlier, fiber counting is perhaps not the way to go, but to evolve procedures and methods whereby workers and contractors will utilize safe procedures to protect workers and inhabitants of buildings at the time that such maintenance and repair activities are undertaken. This requires education of workers, training in safe procedures which are available, enclosures of work areas so that the fibers will not be spread beyond the work region, effective cleanup, safe disposal of the asbestos.

Much has been learned, actually, by the...and much has been developed by contractors in the United States who have specialized in the removal of asbestos in school buildings, and their techniques are very effective in controlling asbestos exposure.

Unfortunately, many removal and many maintenance activities are undertaken by ill-trained and unaware workers with no control and with consequences that can be of considerable seriousness.

So I would commend you to consider mechanisms that would most effectively control this sort of problem, the problem of asbestos that's already in place in buildings and in other areas in Canada and Ontario.

I think that's probably all for the slides.

MR. LASKIN: Thanks, Dr. Nicholson.

Perhaps we can take...shall we take about five minutes, Mr. Chairman?

DR. DUPRE: Why don't we rise until about four-fifteen?

MR. LASKIN: Four-fifteen? Good.

THE INQUIRY RECESSED

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THE INQUIRY RESUMED

DR. DUPRE: Counsel, if I may before we start, if I may try to wrap my arms, so to speak, around the geography of today and tomorrow...

MR. LASKIN: Sure.

DR. DUPRE: It is my understanding that we sit tonight as long as we can, which is no later than six, because of time requirements here...

MR. LASKIN: As long as...

DR. DUPRE: To say nothing of the good will of our witness, who we have already imposed upon to no small extent.

Now, the geography of tomorrow, am I to understand that we could start at nine o'clock?

MR. LASKIN: Again assuming, or impinging upon the good will of Dr. Nicholson.

THE WITNESS: How long do you plan to go tomorrow?

DR. DUPRE: Well, that was going to be my next line. How would two-thirty, at the latest, grab you?

MR. LASKIN: Well, I've canvassed the theme with my friends and I think if we start at nine, they're, of course subject to whatever may arise, but they are fairly confident we could finish everything by one o'clock, if we started at nine.

Now, again...

DR. DUPRE: Are you making a small allowance there for what we euphemistically call the Commissioners' Hour? That's kind of a Children's Hour when we get a chance to ask a few question, too.

MR. LASKIN: I am.

DR. DUPRE: Does that include an allowance of a Commissioners' Hour so that we could be finished by one? Is that agreeable?

MR. LASKIN: Is that all right with you...?

THE WITNESS: That's fine. Sure.

If you were going to go from nine to six, I would raise some objections.

MR. LASKIN: I wouldn't blame you.

DR. DUPRE: I wouldn't dare ask you where you are on your dose-response curve from the dose of us that you've had so far.

Proceed, counsel.

MR. LASKIN: Thank you, Mr. Chairman.

MR. LASKIN: Q. Dr. Nicholson, from what we were discussing before the coffee break, I understand that there is now evidence which would show asbestos-related disease both in the homes of asbestos workers and in the neighborhoods of asbestos plants, manufacturing operations or whatever?

THE WITNESS: A. Yes, that's correct.

Q. I am just wondering whether there are any measurements, any nanogram measurements of those environments against which we could look...?

A. There were no measurements made of the circumstances in which the disease has subsequently...excuse me, the disease has subsequently been related. That is, we are talking about exposure thirty or more years ago.

Q. But do I take it that...?

A. We've looked at home circumstances and we looked at environmental circumstances, and what one can say is that it's hard to imagine a factory as uncontrolled as it might have been in the 1940's and 1950's in the east end of London, sending more asbestos out into the environment than a person on the fortieth story of a skyscraper in New York can

A. (cont'd.) do by blowing it with a hose. So that the only connection one can make in these areas is one of common sense.

Q. Could I take you to page forty-five of tab number nine, your book on dose-response.

A. Okay.

Q. You there have, as I understand it, some asbestos air concentrations in various environments?

A. Right. They are the concentrations we were just talking about.

Q. I then note from nine to nineteen hundred and fifty in public schools with asbestos surfacing materials, from a hundred to five thousand in the homes of asbestos workers, and from eight to three seventy-five within some number of meters of spray sites, and so on. Do I take it that those, while they may not be particular sites from which there has been disease, they, in your view, would give some rough idea of the kinds of concentrations that one might have been looking at?

A. Yeah, I believe these concentrations are evidence of exposure that would not be...that would be of consequence for later disease, and I think that they are not that different from exposures that would have obtained in past years, from our efforts to find serious contamination. We've looked in a lot of places and the highest values ranged, typically, from one hundred to a thousand nanograms per cubic meter of air.

Q. Can I ask you whether to your knowledge there has been any study of or any evidence of asbestos-related disease amongst school children?

A. No. That's in part because it has never been seriously looked for. In part you have the problem of the kids exposed in one circumstance, moving on and they have not been followed. There have been mesotheliomas documented in individuals who are maintenance employees of schools, but that's

A. (cont'd.) the only relationship that I know of.

Q. Have you had some evidence of that in the United States?

A. Yes, there have been such cases described.

Q. What about any...to your knowledge has there been any study of or any evidence of asbestos-related disease amongst the ordinary population who may, you know, on a day-to-day basis frequent skyscrapers where there is...

A. Again, we don't have definitive studies, and we also have a situation where the exposure has been...is a fairly recent one. That is, the high-rise construction with asbestos fireproofing began in 1958. It reached its peak in the mid-1960's. So we are talking about a time from onset of exposure, at this time, of about fifteen years, on average. The process stopped in 1972.

The likelihood is that we will not see the disease and identify cases for some time to come yet.

Q. Can I turn...

A. Where you can begin to see numbers that would allow you to make an association. You can find, you might find a single case of an individual who worked in a building with asbestos, and one never knows whether this is an isolated case with perhaps some other exposure or whether it is real. It's only when cases begin to mount that you can make causal relationships with certainty.

Q. I take it the Anderson et al work which you showed us before, looked at household contacts, family contacts. Has there been any work by your group looking at neighborhood effects?

A. Yes. There is a study that is ongoing of the effects on the neighborhood residents of that Patterson plant. A preliminary or an initial, publication of initial findings

A. (cont'd.) appears in volume 330. It's Mortality Experience in the Neighborhood of an Asbestos Factory, by Hammond, Garfinkel, Selikoff and Nicholson. This was a mortality study that was conducted through 1976, I believe, of individuals resident within half a mile of the Patterson plant, and compared to individuals resident in another portion of town.

Overall, while there was one mesothelioma found in the group adjacent to the factory, and another case of a person who worked in that area, the overall mortality experience was not that different between the two groups, but recognition should be made of the relatively short followup. We are talking about reaching the thirty-year period from onset of exposure, and largely we would be expecting to find the mesothelioma, expecting mesothelioma to be the cases that would be of concern.

In a study that was done with this and attempting to not measure quantitatively the air concentrations that people were exposed to, but to get a measure of the relative exposure, we analyzed dust from homes of these neighborhood contacts and found that the exposure was largely confined to those that were employed, that lived within a quarter of a mile of the plant, and very little difference could be found in the concentration of asbestos fibers in settled dust in those beyond that quarter mile, compared to those resident elsewhere in the community, in the control area.

So that actually the number of people at risk in the study were somewhat limited, and that also limits its general applicability.

Parenthetically though, in that nearby group, we were finding concentrations of asbestos in settled dust approximately equal to those that we found in the households of the workers' families at that time, so that the potential for risk, at least in the nearby residents, which only constitute

A. (cont'd.) a very small portion of the study group, may be significant.

Q. You are continuing to follow that?

A. Yes, it's an ongoing study. There will be later tracing done sometime in the future.

Q. Can I ask you, just dealing with environmental exposures or not-direct occupational exposures, is it your judgement that mesothelioma is the greater health hazard, if I may put it that way?

A. It is certainly the greater health hazard for individuals exposed at younger years. That is, under age twenty. For adults, it moves towards lung cancer where you have the interactive effect of cigarette smoking, and there it only obtains in those individuals who smoke cigarettes.

But I think while the data are yet to come in and they will only...they are beginning to be available from the cohort study of family members, there is very limited data on environmental disease other than mesothelioma and the finding of x-ray abnormalities, but not on lung cancer at this time.

Q. You mentioned this morning, for example, that the dose necessary to produce an excess risk of lung cancer was one, as I understood it, lower than to produce an excess risk of asbestosis, or death by asbestosis?

A. Oh, right. That's certainly the case.

Q. Can we make any comparisons as between lung cancer and mesothelioma, on the question of dose?

A. It's hard to say, because we just don't have the evidence of the comparison of lung cancer with mesothelioma in the very low-exposure circumstances. The data that we have is largely case findings of mesothelioma, but what would be important to have is the information on the relative risk of mesothelioma and lung cancer in some of these

A. (cont'd.) lower-exposed groups to, in particular, verify the extension to environmental concentrations.

MR. LASKIN: I think the chairman has a question.

DR. DUPRE: I just want to make sure, Dr. Nicholson, that I am understanding this in my own simple way. My understanding at the moment, but please teach me the error of my thinking, is as straightforward as the following: Because mesothelioma is so rarely found within the general population, a mesothelioma death is, shall we say, a visible epidemiological event?

THE WITNESS: That's right.

DR. DUPRE: Whereas a lung cancer death is hidden by all kinds of associated factors, background noise and so on. Would that be a correct layman's understanding?

THE WITNESS: Yes, that's absolutely correct. I should have established that. When you...in order to establish a lung cancer risk at low levels, you need to follow a large population over a considerable period of time in order to distinguish the rate expected with that observed, and this is much more difficult than in the group of asbestos workers highly exposed.

Any time you are looking at differences of rates where the background rate is already significant, you have a hard study in hand.

MR. LASKIN: Q. Just to come back to mesothelioma and this relationship with age, which I understood your evidence this morning...can I ask you whether your group at Mount Sinai has uncovered any evidence one way or the other as to whether the lungs of young children, or the body function of young children, makes them more or less susceptible to asbestos exposures, compared to adults?

THE WITNESS: A. We don't have data on whether there is greater human susceptibility, per se. The thought that

A. (cont'd.) perhaps there is comes from the effects of other carcinogens that are more often manifest at the time that cells divide. That may be the case with asbestos, in which case children per se could be more at risk.

It may not be, since one of the tumors that you are concerned with is pleura, which is not a rapidly-dividing tissue, and I think that at this time we can't say what in fact the case is with respect to that question, but the issue of the time dependence of mesothelioma certainly makes children especially vulnerable.

Q. Can I turn to schools directly, and just a few questions? As I read through your major New Jersey study, it seemed to me that one of the issues you looked at was the efficacy of the various control techniques that might be available, and I'm wondering...it may be a little outside the scientific realm, but I would certainly be interested in your professional views as to the merits in various situations as between removal on the one hand, and sealing on the other... or indeed simply enclosure, if that's a fair alternative.

A. Yes. This study was actually undertaken because of the...initially the desire of NIAHS to see if there were...to look at the efficacy of sealing methods from what would appear to be a desirable economic factor in their favor, and we looked at...we did two jobs where that was done. One was a total disaster in that the material, the sealant material, was sufficiently inflexible that when...that it ended up adding weight to the friable material and cracking it and pulling it down, so it had to be removed.

The other one was applied to semi-cementitious material, that is the vermiculite matrix, and circumstances were there was already a substantial substrata, and that was effective.

But what...we found that certainly with friable asbestos, sealants were not a particularly effective control

A. (cont'd.) measure.

What has subsequently developed with much more experience by a wide variety of people, is that sealants are very ineffective in general, and that largely the method of choice is to remove the asbestos. You get the problem controlled for all time. You don't have to first worry about the integrity of the sealant being maintained through damage and age, building use, water leakage and all the other possibilities that can happen. You don't have to worry about the problem that if something does go wrong that you've got a much greater removal problem in trying to deal with now a material that has been sealed and which precludes effective wetting. You don't have to worry about the problem of what to do with the damn stuff when you end up having to take the building down.

So, if for a slightly greater cost...and in some cases the cost difference is very little...you can get rid of the whole situation and remove the material safely, that's the option that one would take. The removal procedures are available that would allow it to be done safely and to have the building effectively cleaned after removal so that there is no significantly greater risk from removal than there would be from other containment mechanisms.

In the estimate of people that have now been engaged in many of these activities is tha t roughly ninety percent of the circumstances...in ninety percent of the circumstances they would opt for removal rather than encapsulation or enclosure. Enclosure is a fairly expensive procedure and again, while you can, you have less of the problems than you do with sealing, in terms of you don't have the added weight, you don't have the problem of trying to wet the material with a coating on it afterwards, you still have the problem of removing that material at a later point in time. So unless there are very extenuating circumstances, I would

A. (cont'd.) strongly advocate removal for open surfaces. The issue...and this is often the case with schools, you are dealing with auditoriums, hallways, classrooms, where it's easy to just seal it off and go over and take the whole thing out. Where there is a real problem, and the answer to the problem is not fully in hand, is in buildings with the air supply system passing through a plenum sprayed with asbestos, and the asbestos being sprayed on a highly-convoluted, complicated system of girders and steel work. It isn't easy to scrape that stuff off and it isn't easy to remove it without having an extremely difficult and costly time. It's also not easy to seal it, although sealants there are not likely to be subject to abuse as they would be on an open surface, so that's one advantage. But the need is to have a sealant that would, that one could be assured would provide greater adhesion to the underlying surface than now exists with the material itself, because if you simply just add a coating material, an added weight, you are going to lose in the end.

What is really needed I think, in the case of fireproofing material in public buildings on material behind hung ceilings, is a really careful look at the control options, and I wouldn't at this point hazard a guess as to what might be the best way to go, but it's a problem that hasn't yet been studied by any organization. There is certainly a need to do so.

Q. Did you, in the New Jersey study, did you look at the kinds of exposures that your maintenance workers, or the maintenance workers who went into the schools to perform whatever control technique it may have been, were subjected to?

A. In that study we described the control measures that were utilized for the workers that either sealed or removed the material.

Q. Yes?

A. You are speaking about those workers?

Q. Those workers, yes.

A. There it was possible with total enclosure of the building...that is, the area to be worked upon...with plastic on the walls and ceilings, and a shower facility nearby, still within the closed areas, and there in the buildings you can usually set up such a shower facility in a washroom or janitor's closet in some cases...some contractors actually have a van that they will back up to a building, and have a shower in it so that an external...the workers go through a door out of the school and into their changing and cleaning facility... with washing down afterwards we could measure no excess concentration with those control measures. Nor could we measure by fiber counting any level in areas of the school that were monitored during the work activity within the double-enclosed area.

Workers, because of the opportunity of adequately wetting the material where you had it being manipulated only while wetted with a surfactant-containing water, there was not a need to have air supply respirators and the workers could use a half-face mask respirator. The concentrations within the area did not exceed two fibers per millilitre in virtually any of the cases that we looked at, because of the effective wetting that was possible, and thus there was not the need for the cumbersomeness of an air supply system, and the workers were protected.

MR. LASKIN: I see the chairman looking at me. No, that's great, Dr. Nicholson.

DR. DUPRE: Forgive me, Dr. Nicholson, but this simply is time at the Commissioners' Hour tomorrow that I will not take.

What you have been orally describing is fascinating to me, and indeed it summarizes very well what you have in one of your articles on the subject, but would you

DR. DUPRE: (cont'd.) please indulge me to look at that text with me, briefly?

THE WITNESS: Yes.

DR. DUPRE: I'm referring now to tab six.

THE WITNESS: Yes.

DR. DUPRE: I am at page thirty-three.

THE WITNESS: Yes.

DR. DUPRE: My first question is a very simple one. The opening sentence under Containment Techniques,

"The work of the contractors was closely supervised and each followed similar general guidelines".

THE WITNESS: Yes, we had a man on the job, on every one of these jobs.

DR. DUPRE: That is my question, the work of the contractors was closely supervised by whom?

THE WITNESS: By...

DR. DUPRE: A member of the Mount Sinai...?

THE WITNESS: A member of the Mount Sinai group, who had...actually it was Ed Swazowski, who had worked with Bob Sawyer from the very beginning of his work going back to the Yale removal experience, so he had fairly...he was very knowledgeable.

DR. DUPRE: Extremely experienced.

THE WITNESS: Yes. And that's an extremely important aspect. It takes a little while for contractors to learn what they can do and to work up their techniques, although one of the contractors that we used...we used three contractors for both sealing and removal. One contractor did two jobs. One of the contractors we had to spend a lot of time with the guys, because his supervisor...while he claimed to have had asbestos experience and we went and looked at some of the jobs they had done, they were really pretty casual. It took half a day before he got everybody into line in doing it properly.

DR. DUPRE: Your paper relates that too, Dr. Nicholson, and that leads me to my second question, which is at page thirty-six, in the bottom paragraph: (sic)

"It was evident during this project that previous experience in removal activities was of benefit".

Then you discuss one contractor's foreman who obviously knew what he was doing. Then you point out...

THE WITNESS: Where are you reading?

DR. DUPRE: Page thirty-five, I'm sorry. Page thirty-five.

THE WITNESS: Yes.

DR. DUPRE: Now coming down to the bottom of that paragraph, having pointed out that one contractor's foreman had considerable experience, you come to the second point, and you then point out and I guess this dovetails directly with the close supervision that the member of your team was giving, you conclude that by saying, "Here the initial work went slowly and much instruction had to be offered by Mount Sinai personnel concerning requisite control measures and removal procedures".

Now you point out, "Once understood however, the workers soon became proficient and the work proceeded smoothly".

Your concluding sentence is, and this is where you were waxing constructive: "This points to the desirability and possible effectiveness of training courses in asbestos removal techniques".

THE WITNESS: I think I could have said it even stronger.

DR. DUPRE: Indeed, I was going to say that I can't help but agree with that conclusion, but am I correct, now that I understand the context, that what was going on here was

DR. DUPRE: (cont'd.) giving that second foreman considerably more than the benefit of a course that he might have taken?

THE WITNESS: Oh, yes. It was very hands-on.

DR. DUPRE: You had a hands-on individual saying, this is how you do it, and the moment he does the wrong things says, no, you do it this way. So there was, in fact, very considerable supervision involved in every phase of this study?

THE WITNESS: That's correct.

DR. DUPRE: Thank you, Dr. Nicholson. I just wanted to make sure that I had understood that, and as such, therefore, the effectiveness of a course is pointed to as one of any of a number of...

THE WITNESS: Yes, but what I meant there is that there would be the opportunity to actually, in that experience, have a mockup room, or something, so that you are not just just dealing with lectures on the board, you are dealing with the operation itself. That's the only way that you could do it.

DR. DUPRE: I realize that, but as an old professor I'm also aware that even with a live demonstration the graduate does not necessarily go out and immediately apply it.

I'm sorry. Thank you for your indulgence, counsel.

MR. LASKIN: Quite all right.

MR. LASKIN: Q. I suppose the only other question that I really wanted to ask you, Dr. Nicholson, about schools, is your view on whether there is any utility in doing counting within schools, whether it be air concentration counts or fiber counts or whatever?

THE WITNESS: A. I don't think there is any utility for doing it in the evaluation of whether one should

A. (cont'd.) undertake remedial action in that school. So that what I'm saying is, in deciding to remove, to enclose or to deal with a perceived asbestos problem, the judgement that is made on the basis of the appearance of the material and of potential exposure circumstances to the kids, is the judgement that should obtain. Because here you are looking at what has happened in the school, you can see the damage that has occurred. Clearly at the time of that damage kids could have been exposed, or they likely were exposed, and the possibility of it taking place in the future exists.

If you...and the degree of damage, the conditions and the general circumstances would suggest the priorities that should be utilized in that particular circumstance. This is a judgemental issue, as in any of these circumstances, whether one has fiber counting data or not.

Now, thus to decide to do something, it is not necessary that one have...firstly, it's not necessary to have air concentrations. Secondly, they can be very misleading because what you are doing is you are sampling over a limited period of time, and you could have a count done at a time that, in which previously very little had happened to that building to disturb the asbestos, so you get a fine reading and say it's okay.

The next day, somebody comes by and it's all over the place, and you are not there to measure it. So the sampling can at best only be a catch-as-catch-can basis, and to have a monitoring program that would allow you to effectively determine what the average concentrations would be, would be of enormous costliness. Because first of all, it would have to be done with electron microscopy, and it would have to be done often, and you would have to do outside background samples, and so on and so forth. It wouldn't provide any more information than you have by looking at something with your common sense.

Secondly, if the program utilized optical

A. (cont'd.) microscopy, it could even be misleading because the fiber concentrations that we are talking about are really below the capability of optical microscopy to provide a meaningful measure, except in the cases of extreme concentrations for environmental circumstances...that is, to the concentrations of a tenth of a fiber per millilitre or higher.

There, too, it's a catch-as-catch-can basis as to whether you would detect such circumstances, so I think no person who has had any experience in this field would advocate that, as a general rule, one would..should take action based solely upon environmental measurements. There's some cases where you might want to investigate general circumstances to see what the circumstances are, that's what we were doing in New Jersey. We were getting a feel of what the possible range of exposures were. We weren't using that for control purposes.

So I'm not belittling analysis, I'm just saying that the judgement for control action should not be based upon it.

Q. Let me just ask one more final question on schools, and it came back to a comment you made earlier about removal versus the use of sealants. I understood, I believe, what you just said. One of the things I thought I had got out of your paper was, that depending upon the type of asbestos application, whether it be loose matrixes as opposed to the cementitious mix, that sealants as opposed to removal might be just as effective. Did I understand you just to say, and I'm sorry I didn't follow it up at the time, that your subsequent analysis has shown, perhaps, that sealants haven't even been quite as effective with the cemetitious mix type of application?

A. I think in the cementitious stuff it's not that bad a circumstance. That's where you've really got a plaster-like material that has fairly great integrity and you are

A. (cont'd.) only concerned with surface brushing of the material.

I think the intermediate circumstance is probably okay, but if it could be removed, even removal might be desired there. That's one which is kind of best described as being kind of a compactible, semi-cementitious stuff. That is, it was a slurry that was applied and it wasn't one of these loose, blown-on materials. There we found it was all right, but the possibility of serious damage to that material still could come. That is the picture that I showed on the slide of that, and I think the picture is here as well. Oh, you can't see it.

Well, if you look at figure five, you'll recognize it...which actually showed a bunch of dents in this ceiling and they came about because the school was once a middle school. It's now...they changed the mix of the kids so they are now largely younger kids rather than eighth and ninth graders, and I forget how they changed the school system, but the older kids in the school previously participated in a drum and bugle...no, a marching band operation for the football team and they had a flag carrying and manipulating crew that would practice their flag twirling in this area, and the flags kept hitting...which happened to have a three-pronged end to it, and the flags kept bumping into this material. That's where much of the damage occurred, although some kids were carving their names in it as well.

But, you know, that very heavy abuse could still occur with sealants, so that that's why the experience of others is not adding to the concerns that we expressed here with respect to the friable stuff, that maybe even some of these circumstances as well, and would probably suggest an even stronger admonition than is contained in here, towards removal than sealing.

Q. Does...is it your judgement that when you are dealing with the loose, friable stuff that removal is the optimum control technique?

A. There is really very few circumstances that would suggest otherwise.

Q. What might those few circumstances be?

A. Perhaps some asbestos in a boiler room that nobody ever goes into, or something like that. Certainly there should be no pupil access, and the material would have to be very well applied and not deteriorating of its own account.

You know, asbestos in a boiler room is of lesser concern where it is not deteriorating and creating the likelihood of being carried out into the pupil-use area. I think when you are talking about pupil-use areas, I can't think of anywhere it would be desirable not to remove.

If you don't remove, then I think the only alternative procedure is to use enclosure - that is, build a permanent barrier underneath it. But that, as I mentioned, can have problems when you get leaky roofs. You get everything else associated with it.

Q. Can I turn back from schools just to the one final topic that I wanted to cover with you, and it relates back to occupational exposures? It's really the question of first of all, early detection of asbestos-related disease and second, arising out of that, what effect, if any, there may be from removal of workers from the workplace.

I'm just wondering whether your group at Mount Sinai has looked at either or both of those questions, and with what results?

A. I think in early detection of disease you are looking at abnormal x-rays. By the time you see an abnormal x-ray, which may come twenty...say it comes at twenty years...the exposure may be really significant, and even in the absence of

A. (cont'd.) further exposure, by thirty or forty years the guy really has serious lung problems, with the exposure also being sufficient to produce a very high risk of cancer. And it's too late, it's wrong to wait until you see that sort of manifestation before doing something.

Removal from exposure certainly will benefit that person and will reduce his risk by the extent that he doesn't get any further exposure.

Unfortunately, it puts somebody else in that same job, to become exposed, and in terms of human disease does nothing. So the issue really is, not looking for disease in workers exposed to asbestos, but removing asbestos from workers so they don't get disease.

Q. I understand that point, but do I take it from the work of your group at Mount Sinai that the most sensitive early detector, generally speaking, has been the abnormal x-ray as opposed to...?

A. I'm sorry. Yes. It's found to be more indicative of an exposure than pulmonary function tests. We find, at least at a statistical level, in populations exposed to very low concentrations significant changes in x-ray before we find significant changes in pulmonary function that we could attribute to that exposure.

Q. Has your group specifically looked at whether you are able to reduce the risk, slow down the progression of disease, from taking people out of the workplace once they've got some evidence?

A. I would go back to the tenet, taking out the workplace is, I think, the wrong question.

Q. Removal from asbestos exposure?

A. No, no. What I mean...the reason I just gave, that you still have the work situation.

A. (cont'd.) What I think we are beginning to look at, and which I think it's important that it be looked at and hopefully there may be some possibilities there for individuals who have had exposure, is a study of firstly the possible identification of ways that one might identify individuals at particularly high risk among those equally exposed. In other words, long-term insulation workers or individuals exposed in other circumstances to asbestos, with that exposure already having taken place...what means exist to predict who would be likely to get cancer and who would not. If a prediction can be made, what might be done there?

The possibility, for example, of first doing this by measurement of immune competence exists. It is certainly known that when one...that in individuals whose immune competence has been altered by virtue of drugs, as in kidney transplants or otherwise, have a particularly high risk of cancer. We have also found in individuals with mesothelioma that in a very few who have an immune system that is normal, that their longevity after identification of mesothelioma can be significant. In some cases individuals have lived as long as nine years, and then either because the disease rapidly progresses or perhaps because the immune system fails, but with a decreased immune competence at that time mesothelioma then spread rapidly and the individuals died.

But the various possibilities from animal studies and for other, that the immune system is a very important aspect in carcinogenesis in humans has led us to undertake a study of that, among other things, of that feature in long-term insulation workers. Firstly to see if it is predictive of later cancer in people equally at high risk, secondly to see if stimulation of it to allow it to become normal again will reduce any possible increased risk from reduced immune competence, and

A. (cont'd.) even to see if prophylactic measures in those that have immune competence might reduce the risk of later cancer, and one is looking at various such drugs as might be appropriate here - thymocin, perhaps even interferon may be of some use here. These are things that are possibilities in the near and distant future.

Other mechanisms of detection, mutagens in the urine, which may be increased in individuals who are able to... who are at lesser risk because they are able to excrete carcinogenic agents within their body, associated with cigarette smoke, for example. Or maybe those that have high mutagens in the urine are at particular risk because they are metabolizing something to a carcinogenic agent. We don't have the information of the significance of such things, and that study is being undertaken to try to determine that. So that possible alteration of what would normally be the grim picture of people already exposed can be undertaken.

Q. Just a final question. What is the...could you tell us what the current state of the medical art is at Mount Sinai with respect to treatment of asbestos-related diseases?

A. I'm not really an expert on that, but I can, I think, summarize it as pretty grim. There is...even though we have a treatment program for mesothelioma, it does little more than prolong a very miserable life for individuals entering it. The treatment itself is not pleasant, as well as the disease.

Lung cancer, the prospects are as good as the prospects for surgery for lung cancer in other than asbestos-exposed individuals, and that's not very good. The early detection techniques have not proven that effective in significantly increasing longevity in individuals, especially at high risk with long exposures, cigarette smokers, if they

A. (cont'd.) have x-rays at least quarterly and sputum cytology, you might pick up the tumors early enough to have some benefit, but it has to be a very major program undertaken in order to do that. You can not do it on a half-year or a yearly basis. That's kind of like whistling in the wind, because by that time, on average, you are going to be too late. You will do a little better if you do it as often as quarterly... but I'm only speaking of people with particularly high risk - older insulators who smoke, or factory workers who smoke, because there the considerable radiation you would be giving and the considerable trouble that would be required of this program would be worthwhile, whereas it is not in a very large population of people exposed at very low levels. I mean there the advantages are not present.

Q. And asbestosis?

A. In asbestosis there is an awareness of the disease and the knowledge of the extent of the disease, and the knowledge of the physiological competence, the pulmonary competence of the individuals by the individual's physician is very important. The fact is that most people with asbestosis die of something else, an intercurrent respiratory infection, and thus if the individual develops a respiratory infection, he should be aware of the seriousness of it for his particular case, as should his physician, in that it should be immediately treated as a medical emergency. Whereas if you and I get a respiratory infection, you just kind of hang in there and it goes away and our pulmonary reserve capacity is so great that we don't have to worry about it. But for somebody who is on the brink with asbestosis and significantly reduced pulmonary function, it really can be the thing that tips them over. So a very close surveillance by his physician is of very great importance, and they can, with effort, maintain a relatively long livelihood, although it's certainly not one that would be

A. (cont'd.) a very active one.

Q. I take it there you are treating whatever other effects there are...

A. You are treating whatever other effects.

Q. You are not treating the asbestosis directly?

A. Yes. There is not much that you can do with the asbestosis itself once...in some cases if an individual has a thickened pleura like was seen on that slide, there are now operations that can be done where that is removed and give relief to the individual, but in terms of parenchymal fibrosis there is not much that can be done. You just don't want to add to the assault that is already there.

MR. LASKIN: Dr. Nicholson, thank you very much. You've been most patient as I've been trying to wend my way through Epidemiology 101, as my chairman likes to call it.

DR. DUPRE: May I now just ask you and the parties, counsel, whoever may feel confident that if we rewarded Dr. Nicholson by giving him time off now, we could get through between nine and one tomorrow?

MR. LASKIN: I'm sure we could, Mr. Chairman.

DR. DUPRE: Miss Jolley? Yes?

Well, on that happy note, may we wish you a good evening and thank you very much for what you've done so far.

We will now rise until nine a.m. tomorrow.

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THE INQUIRY ADJOURNED

THE FOREGOING WAS PREPARED
FROM THE TAPED RECORDINGS
OF THE COURT PROCEEDINGS

Edwina Macht
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